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PORT PHILLIP'S EARLY DOCTORS, 1835-1839.[1]

By G. T. HOWARD, B.A., M.D.,
Melbourne.

NOT so very long ago my knowledge of medical history was probably not more extensive than that of my contemporaries who had kept abreast of the times. I was not sure whether Æsculapius was a divinity or deified man. I knew that Hippocrates and Galen were medical giants in their own days, and then I realized that medical practice, not science, had been hibernating for over a thousand years. Of English names Harvey and Sydenham were most familiar, then those original geniuses of the eighteenth century, the Hunters, Jenner and Priestley, and so with slightly increasing knowledge to the present day.

When the time of increased leisure arrived, I became a member of the Historical Society of Victoria, where I listened at first with rather languid interest to contemporaries reading the diaries of their grandfathers in the thirties and forties of last century. As time went on my interest quickened, and I began to delve amongst old Victorian medical records, and later on among those of the old and new worlds. Mere interest gave way to fascination, which increased as time went on, and I found myself, somewhat to my surprise, writing and lecturing on the subject. And so, when I received a request to deliver the initial address before this youngest of the sections, I took it as a graceful note of appreciation of my work, and in addition, a greatly esteemed honour.

As the subject was specifically named for me, my only difficulty was where to begin and where to end. During the first five years, 1834-1839, of settlement in Port Phillip there was no direct communication with England. Settlers came from Launceston in

[1] Read at the inaugural meeting of the Historical Section of the Victorian Branch of the British Medical Association on November 3, 1933.

small numbers and overland from Sydney in still smaller numbers.

In 1839 assisted immigration came into force, with the result of an addition of 10,000 to the lay population, including all the ships' doctors. If, then, I were to take in the period 1839-1842, I would have to content myself with reading out a long list of names and practically nothing more. So I have decided to restrict myself to the few pioneers who ventured to our shores in the earlier years. But I think no record of those early days would be complete were I to refrain from all reference to some extremely interesting medical birds of passage who alighted and remained on our shores for only a very brief space before the days of permanent settlement.

Pre-Settlement Visitors.

When Abel Janssen Tasman in 1642 discovered and named Van Diemen's Land, as it was until 1853, he assumed that it was part of the mainland, charted it as such, and went on his interesting way to discover New Zealand. Assuming the accuracy of that chart, our first fleet rounded the southern extremity of Van Diemen's Land and landed its cargo of exiles, voluntary and involuntary, in Sydney Cove on January 26, 1788. Its rare successors followed the same course for the next ten years.

In 1795 His Majesty's Ship *Reliance*, with Captain John Hunter, New South Wales's second Governor, arrived at Sydney. Amongst its officers were two, George Bass, surgeon (aged thirty-two), and Matthews Flinders, midshipman (aged twenty-one), who were destined to attain imperishable fame in the roll of Australian navigators. Their first adventurous freak was to pass out through the Sydney Heads in a dingy eight feet long, accompanied by a small boy. They reached and explored Botany Bay and George's River and returned safely. A little later, in a somewhat larger boat, they reached and returned from Bulli after a perilous voyage. Then they attempted to scale the Blue Mountains, but found its hills and hollows more intractable than the nautical ones with which they were familiar. Later on they reached Coalcliff, about thirty miles from Sydney, and were the first to discover South Coast coal.

Then came the great adventure. In the absence of Flinders on another mission, Bass, with six men and six weeks' provisions, fared forth in an open boat on December 3, 1797, to explore the hitherto unsurveyed south coast. He eventually discovered Twofold Bay, rounded Cape Howe and was soon opposite Cape Hicks (Everard), the first Australian landmark observed by Captain Cook. Shortly afterwards he landed to replenish his water supply, and in doing so became the first doctor to touch Victorian soil. He eventually reached the large bay which he named as his terminal point, Western Port, after a voyage of 600 miles, on January 4, 1798. After a fortnight's rest he commenced his leisurely homeward journey, in the course of which he ascended Wilson's Promonotory, the first white man to do so, and gazed upon the magnificent seascape of the Eastern Archipelago. He now felt convinced, though not, of course, absolutely certain, that Van Diemen's Land was an island. On his return to Sydney he was lionized for his wonderful performance, probably never equalled by any but trained seamen like Bligh; and his boat was for long an object of adoration, but eventually disappeared owing to the activities of curio hunters.

Flinders returned soon afterwards (October 7, 1798) and with Bass in the schooner *Norfolk* (25 tons), circumnavigated Van Diemen's Land, proving conclusively the existence between it and Australia of a broad strait, appropriately named after its discoverer. Bass's subsequent history is that of an adventurous South Sea trader, in the course of which he left for South America in February, 1803, and was never heard of afterwards.

The next item is not strictly medical, but cannot well be ignored. The first boat to pass through Bass Strait from England was the *Lady Nelson* (60 tons) in 1800. It crossed directly from Cape Otway to Cape Schank and so missed Port Phillip Heads. On November 21, 1801 (next year), Lieutenant Murray was sent in the *Lady Nelson* from Sydney, without a doctor, for the express purpose of filling in the gap in the chart between Cape Otway and Cape Schank. Three months afterwards, February 15, 1802, the *Lady Nelson* entered Port Phillip Heads, anchored off Sorrento and did not go further north than Mornington. During a stay of nearly a month Lieutenant Murray did nothing in particular and did it very well.

On April 26, 1802, six weeks after Murray's departure, Matthew Flinders, now captain of the *Investigator* (360 tons), outward bound from England, entered Port Phillip Heads and anchored at Sorrento, at first under the impression that he was in the Western Port (which he had not yet seen) of his friend George Bass. Within twenty four hours he had ascended Arthur's Seat, near Dromana, whence he saw the real Western Port less than twenty miles away to the east. Then he thought that he had discovered the great bay afterwards named Port Phillip, and decided to proceed to its northern end. Unfortunately his ship went aground on a sandbank off Mornington. His food and water supply practicaly exhausted, he reluctantly abandoned further northern exploration, left instructions to his second in command to take the boat, after it floated off, down to Portsea, just inside the Heads. Then in the ship's boat he crossed to the west, ascended the highest peak of the You Yangs, took careful note of all he saw, returned to the Heads and thence to Sydney, where he found that Murray had anticipated him in the discovery of the bay. Flinders had in six days done more survey work than Murray had in twenty-six. There is no mention of a doctor in Flinders's log, but there was one there disguised as a botanist—Robert Brown, who at every opportunity had made a study of the local flora. During the next three

years Brown made exhaustive investigations into the flora of New South Wales and took such voluminous notes that it was not until ten years after his return to England that he published the result—*"Prodromus Florae Novae Hollandae"*. Academic results were F.R.S., D.C.L. (Oxford), LL.D. (Cambridge), similar honours from the Continent, and presidency of the Linnean Society, but material results were microscopic. He survived until June 10, 1858.

The third vessel to enter Port Phillip was the little Sydney built boat, the *Cumberland*, which had a romantic history of its own. During a lull in the Napoleonic wars two French ships, ostensibly exploring, visited Sydney and their crews were courteously entertained. After their departure, Governor King, suspicious of their intentions, sent the *Cumberland*, under Lieutenant Robbins, R.N. (aged twenty-one), to find them and tell them that Great Britain claimed Van Diemen's Land and all the islands in Bass Strait. Robbins found them on the east coast of King Island, told them his instructions, and planted the Union Jack behind their tents, whereon the Frenchmen courteously invited him to dinner. After that, King Island was explored in search of suitable land for agriculture.

Port Phillip was reached soon afterwards (January 20, 1803) and a careful survey of its shores made by Charles Grimes, Surveyor-General of New South Wales; his assistant was Meehan, and McCallum was the doctor of the expedition. McCallum collapsed on the march owing to the heat and remained on board for about a week; but he was with the shore party when on Wednesday, February 3, 1803, the Yarra was discovered. The party remained in the vicinity of the future Melbourne for a week, its farthest east being Dight's Falls, and its farthest north Braybrook. On Sunday, February 7, 1803, they held a picnic at Batman's Hill, followed by a stroll. Owing to the parched appearance of late summer, they were not favourably impressed. After the expedition returned to Sydney nothing more was heard of Melbourne's first medical visitor, Dr. McCallum (his Christian name and qualifications are unknown).

In October, 1803, two ships, the *Calcutta* and the *Ocean*, anchored off Sorrento for the purpose of forming a penal settlement there under Lieutenant-Colonel Collins. There were over three hundred convicts and one hundred others, including officers, crews, marines and free settlers. Amongst the convicts was one Fawkner, whose wife, daughter and son, John Pascoe, aged eleven, were permitted to accompany him. Collins was so very unfavourably impressed with the situation that he asked to be removed to Van Diemen's Land, prophesying amongst other things that "when all the disadvantages attending the Bay are publicly known it cannot be supposed that commercial people will be very desirous of visiting Port Phillip"!

Governor King acceded to his request, and on January 30, 1804, half of the expedition, including the principal officers, left for Hobart, Van Diemen's Land, the remainder following in March. The only items of medical interest in the diary of the chaplain, the Reverend Robert Knopwood, M.A., during the first three months were the first death of a white man (Skillicorn) in Victoria, the birth of Victoria's first white child, a boy named Thorne, one marriage and one death (from gunshot wounds) of an escapee. I have seen it stated that after the departure of the first half there were twenty deaths from scurvy, but I cannot find any definite evidence on that point.

Incidentally one learns that the soldiers were allowed ten ounces of rum per day. Reading between the lines, one infers that the officers' allowance was much larger. Of the doctors there is no mention made, except their mere names, Janson, Bowden, Hopley, Bromley, and that they spent much time in hunting and fishing.

In 1824 Hume and Hovell came overland from Lake George to near Geelong. Their visit is of no medical importance, but their laudatory report encouraged renewed attempts at the colonization, by private enterprise, of Port Phillip.

In 1826 another official settlement without a doctor was established at Corinella, Western Port, and proved, like the settlement at Sorrento, a failure.

Permanent Settlement.

These failures seemed to the Governor of New South Wales, Sir Ralph Darling, such convincing evidence of the unsuitability of the future Victoria for colonization that he refused to allow some prominent settlers in Van Diemen's Land to make a further attempt.

Ignoring this veto, Edward Henty landed at Portland on November 19, 1834, and commenced the settlement of the western district. Early in June, 1835, John Batman, as representative of a syndicate of which J. T. Gellibrand (Attorney-General of Van Diemen's Land) was the head, found himself on a hill near Sunbury, about twenty-five miles north of Melbourne. Thence he saw a wide extent of lightly timbered, well watered and well grassed country, gently undulating and of volcanic soil—ideal sheep country. He hurried eastward about twenty miles, and there made his famous bargain with a native chief, namely, about one thousand square miles of country for £200 worth of trade goods and a small annual payment—a bargain which the Government later on ignored. Soon afterwards he hurried back to Van Diemen's Land with his optimistic report and did not return for five months.

Meanwhile, John Pascoe Fawkner, the erstwhile boy of eleven at Sorrento, was, after a very versatile career, now forty-three, and was just then merely hotel-keeper, newspaper proprietor, bush lawyer and a general nuisance (within the law) to the Government. He organized a rival expedition to go across the straits, but became so seasick that his ship,

The Enterprise (55 tons), turned back, landed him at Georgetown, and went on, arriving in Hobson's Bay towards the end of August, 1835. There was a bar at the old mouth of the Yarra which was narrow, tortuous and full of snags and sandbanks, but the dogged perseverance of these unofficial pioneers ended in the tying up of the Yarra's first ship at "The Falls" at the foot of Queen Street on August 31, 1835. Next day the landing party, Captain Lancey, George Evans, Marr, William Jackson and employees Wyse, Morgan, Evans, Gilbert and Mrs. Gilbert disembarked and erected a sod hut for their gear. That little hut was the foundation stone of marvellous Melbourne, and that little party may be considered the real founders of the city of Melbourne, and, with their little hut, deserve a prominent place in any pageant descriptive of its infancy.

William Jackson soon abandoned the village and led his flock of sheep to near the modern Sunbury on Jackson's Creek, named after him. A few years later he abandoned his holding to W. J. T. Clarke (founder of that dynasty in Victoria) and removed to the Casterton district in the extreme west of Victoria. There was born in the middle fifties his nephew, Ernest Sandford Jackson, who became a Melbourne medical graduate in due time and has for half a century been a leading practitioner in Brisbane and an interesting link between the infancy of Melbourne and the medical profession.

That landing day, September 1, 1835, was also the opening day of a period of thirteen months unique in the history of British colonization, during which there was not to be found in the whole extent of modern Victoria, where settlement was going on rapidly, a soldier, magistrate, policeman, customs officer or tax-gatherer of any kind. How the pioneers dispensed with these indispensables as well as with red tape, sealing wax and pipe clay is another interesting story.

A comparison with the Pilgrim Fathers suggests itself, but I am afraid the unctuous rectitude of the Yarra Fathers was not equal to that pervading the Plymouth Rock. In addition, the cargo of the *Mayflower* on its return to Boston consisted mainly of West African slaves, while the principal item of cargo on the return visit of *The Enterprise* on October 10, 1835, was John Pascoe Fawkner, a little waspish bundle of perpetual motion, destined to play for a generation a prominent part in the public life of Melbourne. He proceeded to put together Melbourne's first house, "the Hub of Melbourne" for about twelve months, a queer architectural contraption about the corner of Flinders Lane and William Streets—Melbourne's first hotel, store, library and club. Before this was completed, Batman, after five months' absence, returned in the ship *Norval* (300 tons) on November 6, 1835, which brought, besides a cargo of sheep and cattle, some young squatters, Cowie, Stead, Steiglitz, Estcourt, and Melbourne's first doctor, Barry Cotter.

Barry Cotter.

The disembarkation at Point Gellibrand (future Williamstown) over, the passengers were rowed up the Yarra, landed, like the Fawknerites, near the Falls, and proceeded to make a night of it drinking one another's health with musical honours.

Barry Cotter, L.F.P.S.G. (no triples then), aged twenty-six, son of a south of Ireland magistrate, had been in Van Diemen's Land since 1830 as government medical officer at Campbelltown, whence he had come over to act as deputy for the Gellibrand-Batman syndicate until they could settle on their allocated areas. There were not in the little village a score of settlers, so medical practice was negligible. For the next five months he was the only doctor between Yass, New South Wales, and Albany, Western Australia, 2,000 miles away. To the settlers he was the unofficial superintendent. He set up a wattle and daub hut on the site of the present English, Scottish and Australian Bank (Collins and Queen Streets), surrounded by the mia mias of his retinue, a dozen natives of Van Diemen's Land. The only interesting medical items recorded before the end of 1835 were the birth of Melbourne's first baby, John Melbourne Gilbert, in December, and a Christmas dinner of fish, fowl and mutton with the official enemy, J. P. Fawkner, following a quarrel with Henry Batman.

In April, 1836, John Batman finally returned from Van Diemen's Land and settled at the lower end of Spencer Street, where he lived until his death in 1839. With him came, amongst others, Melbourne's second doctor, Alexander Thomson.

On September 30, 1836, Melbourne's period of idyllic and possibly boresome anarchy was ended by the arrival from Sydney of law and order in the persons of Captain Lonsdale and a retinue of about eighty, including thirty convicts, thirty soldiers, three policemen, three surveyors; hardly any teetotallers. He reserved for departmental purposes a block in the west end bounded by Spencer, Collins, King and Bourke Streets, and took a census, from which it appeared that the most important citizens were John Batman, J. H. Wedge, and the two doctors, Thomson and Cotter, the latter of whom was the owner of 2,500 sheep, which, with Batman's, were grazing over the west, north and eastern areas of the future city. (Fawkner had returned to Van Diemen's Land, where he remained for nearly a year.)

In January, 1837, Cotter was appointed Government Medical Officer, *vice* Thomson, resigned.

On June 1, 1837, at the first land sale, he bought the Leviathan corner half acre, south-west corner of Bourke and Swanston Streets, for £30. Later on he bought Alston's corner (north-east, Elizabeth and Collins Streets) for some small amount and became part proprietor of the Lamb Inn Hotel on the site of the present Scott's (these properties are now worth £1,000,000).

In 1838 he fought the first duel in Melbourne with the quarrelsome editor of the first printed

newspaper, George Arden, without injury to either party. In the same year he married in Launceston Miss Fitzgerald, from Limerick, by whom he had a family of four—two sons and two daughters. His name appears in the contemporary newspapers frequently in connexion with various business propositions; only twice with medical matters. On the first of these occasions he reported scurvy amongst the prisoners, and the second is an advertisement announcing that he is in business as doctor and chemist and has for sale a variety of delicacies, from sago to turpentine, arrowroot to spirits of tar, with candied lemon and bluestone, lemon syrup, corrosive sublimate *et cetera*.

But his heart was more in sport and business than in his medical work. The promoters of the first Melbourne Club (at the corner of Market and Collins Streets) held preliminary meetings at his house. He also kept racehorses and was secretary and treasurer of the first race meeting at Flemington in March, 1840.

About that time Melbourne's first financial boom was at its height. He had evidently been very successful, and publicly announced his retirement from the profession prior to a long visit to Europe. Before leaving, he took medical charge of the ship *Glen Huntly*, quarantined at Point Ormond with typhus patients, and stayed there until the trouble was over. On his return from Europe the boom had collapsed and he was practically ruined. His wife and family left him because of his tendency to intemperance. He departed for Europe in the *Isabella* in 1844. The captain was insane and Cotter deposed him soon after leaving the Heads, but it was too late—the ship was wrecked on Chappell Island, Bass Straits. He returned to Melbourne, thence to Sydney in 1845. Until 1865 he cannot be traced; then he was successively at Menindee (on the Darling), Wilcannia, Wentworth, Balranald, Swan Hill, where the end came in November, 1877, at the age of sixty-eight.

Alexander Thomson.

Melbourne's second doctor arrived in the *Caledonna* in April, 1836, from Van Diemen's Land. He was thirty-six years of age, a native of Aberdeen, and had come out to Van Diemen's Land in his own ship. On arrival in Melbourne he became medical officer to the Batman syndicate at £200 *per annum* and resided near Saint Paul's Cathedral. He rode down at an early period to the site of Geelong and took up, as its second settler, some land near the Barrabool Hills and put a caretaker in possession. Later he drove a bullock dray down with employees, but his men ran away at the Werribee, leaving him to go on alone.

When Captain Lonsdale arrived on September 30, 1836, he made Thomson Government Medical Officer, a position which Thomson resigned in favour of Barry Cotter in January, 1837.

His after history is for a generation that of the grand old man of Geelong—its first doctor, mayor, newspaper proprietor and Presbyterian elder.

With Learmouth, Yuille and some others he first ascended Mount Buninyong and then, crossing the site of future Ballarat, went on to the site of Learmouth with them.

At the first land sale he bought Hosie's corner, Elizabeth and Flinders Streets, for £46. He devoted a great deal of time and energy to public affairs, being a determined opponent of the admission of convicts to Port Phillip and an equally warm advocate of separation from New South Wales. When the parent state became possessed of a legislative council, he was one of Victoria's first representatives in 1843. Later on he represented Geelong for a short time in the Victorian Parliament. His daughter married Mr. Henry Creswick, the founder of a well known pastoralist family. Full of years and honours he died in 1866, aged sixty-six.

Patrick Cussen.

A regimental medical officer, Patrick Cussen, aged forty-five years, came over from Sydney in an early steamer, the *James Watt*, in September, 1837, and took over from Barry Cotter, at a salary of 7s. 6d. per day (£137 17s. 6d. *per annum*), with £50 5s. for house allowance. He lived in Market Street at first, then on the site of the present Law Courts until his death in 1849. The hospital accommodation for soldiers and prisoners was very primitive and he carried on there with great difficulty. He performed the first recorded operation in Melbourne (amputation of an arm) in 1839, due to the bursting of a blunderbuss. He also amputated a penis for "venereal"; no details are given. He was first President of the Port Phillip Medical Association in 1846 and an early member of the Melbourne Club. The chronicler says he was dubbed "Old Cussen" and was a rosy-cheeked, fussy, popular, efficient government official. His step-grandson, Mr. E. C. O. Howard, born in April, 1839, is now a Melbourne resident in fair health.

Edgar Lovell Byass.

Edgar Lovell Byass was one of the first doctors in South Australia at Kangaroo Island in 1836 and first doctor in Portland, Victoria. His daughter was first white girl child born there in 1838. In the latter district he lived partly as pastoralist and partly as doctor until 1874.

John Sproat.

John Sproat, L.R.C.S. (Edinburgh), 1824, M.D. (Edinburgh), 1828, arrived in Melbourne in 1838 or 1839 and left finally for Scotland in 1854. He was evidently an unobtrusive person, as there is practically no other record of his presence. He married a Miss Langlands, thus becoming the uncle of a late colleague, F. H. Langlands, F.R.C.S.

Jonathan Clarke, M.R.C.S.

Jonathan Clarke, M.R.C.S., was Gellibrand's manager at Lara in 1836, then became Geelong's first Government Medical Officer.

David John Thomas.

David John Thomas, M.R.C.S. (1838), L.S.A. (1838), aged twenty-six, arrived in Melbourne in May, 1839. He was the only one of the group who had filled the post of resident medical officer in a British hospital. He settled in Bourke Street West, opposite old St. James's, and remained there until 1853. During the intermediate period he was the undoubted leader of the profession. In addition, he was an incorrigible practical joker and the hero of a hundred stories.

In 1840, with his colleagues, Wilkie, Myers and O'Mullane, he established a stop-gap general hospital in Bourke Street West, which carried on until the opening of the Melbourne Hospital in 1848. In August, 1847, he administered ether (the first to do so in Victoria) and then amputated an arm under somewhat dramatic circumstances. He was also one of the first to administer chloroform in 1848. He was the first in Melbourne to perform under anæsthesia operations such as ligature of the common carotid and femoral arteries, and excision of the jaw.

In 1847 he was elected as one of the first surgeons to the Melbourne Hospital, his colleagues being W. H. Campbell and A. F. A. Greeves.

He married Miss McCrae, thus becoming the uncle of G. G. McCrae.

From 1853 to 1859 he was in Europe, where he acquired the extra qualifications of F.R.C.S. (England) and M.D. (St. Andrews). On his return he lived in Collins Street and again became surgeon to the Melbourne Hospital. He contributed to the *Australian Medical Journal* a series of valuable original articles. He became President of the Medical Society of Victoria in 1865.

After a very full and useful life he died suddenly in 1871. One daughter (now aged eighty-three) survives him.

One of the numerous stories handed down about him, possibly with some embroidery, is the following. After a festive night he realized he could not reach home without help, so, hailing one of the few cabmen of the forties, the following colloquy took place.

T.: Cabby, do you know Dr. Thomas?

C.: No, Sir.

T.: Do you know Dr. Wilkie?

C.: No, Sir.

T.: You surely cannot have been here very long, seeing that you do not know these prominent gentlemen.

C.: Only been here a fortnight, Sir.

T.: Do you know where Dr. Thomas lives?

C.: Great Bourke Street West, opposite St. James's Cathedral.

T.: Do you know where Dr. Wilkie lives?

C.: At the corner of Swanston Street and Flinders Lane.

T.: Well look here, Cabby. I am Dr. Wilkie, and if I went home in this condition you know what a row there would be—married man yourself, I suppose. Drive me to Dr. Thomas's and I'll stay there for the night.

Arrived there, T., in a fuddled way, felt in vain in his pockets for the fare, so he said at last:

I have no money, Cabby; you come to my place in Swanston Street tomorrow at 11 o'clock and I'll pay you double fare.

Next day at 11 a.m., Dr. Wilkie himself, a dour Presbyterian elder, opened the door to the cabman. Further comment is unnecessary.

E. C. Hobson.

E. C. Hobson, M.D., the only Australian on this list, a native of Parramatta, educated in Tasmania and subsequently in England, came to Melbourne in 1839. He had a flair for zoology and was thought highly of by Sir Richard Owen. Like many others, he owned a pastoral property near Frankston. He was elected physician to the Melbourne Hospital in 1847, but died very soon afterwards (in 1847) before its opening. His popularity was such that a monument was by public subscription erected over his grave in the old cemetery.

George MacKay.

George MacKay, father and grandfather of two popular medical representatives of Clan McKay, came to Myrrhee in the Ovens Valley in 1838 and sold out in the early forties to Dr. (afterwards Sir) Francis Murphy, who was first speaker of the Victorian Legislative Assembly in 1856.

David Wilkie.

The last on the list was a Scotchman, a good representative of the old family doctor, a stickler for the conventionalities, a Presbyterian elder, and for a long time a member of the Legislative Council. His friend Thomas played many a practical joke upon him. One of his pet antipathies was the proposed Yan Yean supply of water, opened in 1857. He predicted a great deal of trouble and, strange to say, there were at first a few cases of lead poisoning. One of his sons, Dan, was a well known solicitor, and in his early days a good East Melbourne cricketer. Another was a medical man (David Elliot Balfour Wilkie). Dr. Wilkie survived all the other pioneers until 1885.

Conclusion.

There is no documentary evidence of the kind of treatment followed by this group, as there was no newspaper until 1838, and no medical society until 1846. With some it is probable that a knife or two, a needle or two, and a skein of horsehair in the waistcoat pocket formed their complete surgical outfit, while calomel (magnesium sulphate had not yet arrived) was almost a panacea for their medical work. Altogether one may imagine that in those pre-anæsthetic, pre-aseptic, pre-teetotal days the patient's lot was not a happy one. And yet these doctors were an interesting band of adventurers, fit companions for their lay brethren, who, after severing themselves from the old world, had, ignoring official veto, come here and carried on in the future Victoria one of the most successful non-official experiments in colonization the world has ever known.

THE DETECTION AND ESTIMATION OF α-DINITROPHENOL, A NEW DRUG FOR THE TREATMENT OF OBESITY.

By ADOLPH BOLLIGER, Ph.D.,
From the Gordon Craig Urological Laboratory, Department of Surgery, University of Sydney.

THE preparation known as 2:4 dinitrophenol or α-dinitrophenol is a yellow crystalline compound obtained by direct nitration of phenol. It is slightly soluble in cold water, but considerably more soluble in the form of its sodium salt or in boiling water. Its solubility in the usual organic solvents is greater again.

Although this phenol derivative was used on a large scale in the manufacture of dyestuffs and explosives before 1914, its peculiar toxic properties were apparently first observed during the world war by French physicians. Many munition workers exposed to dinitrophenol complained of general weakness associated with sweating, headaches and dizziness. Others, in the course of exposure lasting for months, lost considerable amounts of weight with, however, no other grave symptoms. After the intake of large amounts death was found to occur within a few hours without any characteristic lesions in the viscera.[1]

Very recently the pharmacological properties of α-dinitrophenol were investigated independently and almost simultaneously in France[2] and in the United States of America.[3] It was found that this chemical greatly stimulated metabolism in animals. The administration of therapeutic doses to a fairly large series of patients showed that the metabolism could be increased by 50% or more over considerable periods of time without any ill effects of importance.[4]

Such a pharmacological action may be found useful in several abnormal conditions. So far it has only been applied on a large scale to the treatment of obesity and quite favourable results have been reported.[4]

Some homologues and substitution products of α-dinitrophenol show similar pharmacological

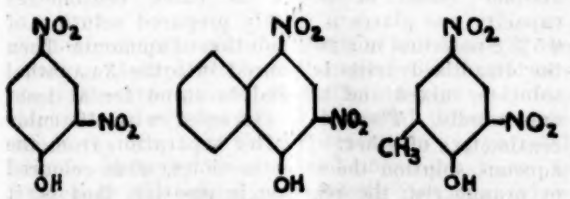

FIGURE I.

2:4 dinitrophenol, 2:4 dinitro-α-naphthol, 2:4 dinitro-o-cresol, compounds of the aromatic series which were found to raise metabolism. Note the 2:4 position of the nitro group in relation to the hydroxyl group in all three compounds.

actions. Heymans, of Ghent,[5] has investigated dinitronaphthol, and Dodds and Pope[6] have recently made a preliminary announcement with regard to dinitro-o-cresol.

If this drug or its homologues should fulfil therapeutic expectations, particularly with respect to the treatment of obesity, it will undoubtedly be used widely by the physician. Undoubtedly also proprietary products will appear on the market containing secretly or openly as their essential basis the low-priced α-dinitrophenol or a similar compound.

Following the indiscriminate intake of this drug diagnostic and even medico-legal problems may arise, and a chemical method of quickly recognizing its presence in medicines taken, or in urine, blood or tissue will almost certainly be of great service. A recent case of acute death following the injudicious intake of α-dinitrophenol for the purpose of reducing body weight is already on record, and this in a physician. The occasional occurrence of skin rashes has also been reported.[7]

The Detection of α-Dinitrophenol.

In work on the titration of methylene blue with 2:4:6 trinitrophenol (picric acid) it has been observed that the 2:4 dinitrophenol forms very readily with methylene blue an addition compound similar to that formed by picric acid.[8] By simply adding a solution of methylene blue to a solution of dinitrophenol methylene blue, dinitrophenolate is obtained, which crystallizes in fine bronze-coloured needles. This compound is sparingly soluble in most solvents to form a green solution. Its solubility in chloroform is greater, but considerably less than that of the corresponding picrate. The practical application of this reaction in the detection of α-dinitrophenol is as follows: The acidified solution is extracted with chloroform and to the neutralized chloroform extract a dilute solution of methylene blue is added. If dinitrophenol or a similar compound is present the chloroform extract will become green.

In examining urine the actual procedure for the detection of traces of dinitrophenol is as follows: In a separating funnel a volume of urine (20 cubic centimetres, if available) is acidified with one-tenth of a volume of 70% sulphuric acid. It is then extracted by gentle shaking for about three minutes with half its volume again of chloroform. If possible, permanent emulsions have to be avoided. However, if after some standing sufficient chloro- does not separate out cleanly, the chloroform has to be separated out by centrifugation. Then about 10 cubic centimetres of the chloroform extract are transferred to a test tube containing a gramme or so of calcium carbonate. The contents are mixed well and 0·0001 N methylene blue, chemically pure (about 0·004%) is added in very small drops. The mixture is well shaken after every drop till the first change of colour of the contents occurs. It is then filtered through a dry filter into another test tube, an equal amount of distilled water is added, it is shaken well again and the water is removed. If the chloroform shows a distinct green colour which cannot be extracted by further washings with water, α-dinitrophenol is present in

the urine. If there is any doubt, some of the chloroform extract may be concentrated by evaporation in order to intensify the colour.

If dealing with amounts of dinitrophenol up to one milligramme *per centum* in urine, the application of the precautions mentioned will be necessary, because some substance present in normal urine in small amounts and extracted by chloroform seems to form with an excess of methylene blue a bluish compound, which may easily be misleading. Therefore, for the detection an excess of methylene blue has to be avoided. But after the presence of dinitrophenol has been established by the typical greenish colour of the chloroform extract, more methylene blue may be added either to get an approximate idea of the amount of the drug present or to perform an actual quantitative estimation by the technique which will be described below. Doubtful results with regard to the presence or absence of the drug may be compared with those obtained with urine treated similarly, which are known to contain no dinitrophenol. With amounts above one milligramme *per centum* the results are beyond doubt.

The detection of dinitrophenol in the blood stream of a non-jaundiced person is more sensitive because the chloroform extract of serum or plasma does not contain a sufficient amount of any substance which reacts visibly with methylene blue. The following technique has been found to be suitable. Serum acidified with a few drops of sulphuric acid is thoroughly shaken with an equal amount of chloroform. Then the mixture is centrifuged and the chloroform extract is pipetted off and filtered through a dry filter into a test tube containing some calcium carbonate. Then 0·0001 N methylene blue is added in small drops till the mixture begins to change colour. After filtering again, the presence of dinitrophenol is indicated by a green tint of the chloroform extract.

In proprictary medicines in which the presence of α-dinitrophenol has been proven, one proceeds as follows for the quantitative determination by titration: A known amount is dissolved in 5% sodium hydroxide. After the solution is acidified with 70% sulphuric acid, the dinitrophenol is extracted with several lots of chloroform. The combined chloroform extracts are treated with calcium carbonate, filtered and made up to a known amount. An aliquot part of the chloroform extract is transferred to a separating funnel and 0·0001 N methylene blue is added from a burette. The technique of the titration is the same as the one described for the determination of picric acid with methylene blue.[8] The methylene blue combines with the dinitrophenol to the chloroform-soluble methylene blue dinitrophenolate. Therefore, on extraction the watery layer originally containing the methylene blue turns yellow, while the chloroform takes on a green colour. The end point is reached when the watery layer becomes colourless. For practical reasons the appearance of the first tint of blue which cannot be extracted with fresh chloroform is regarded as the end point. In other words, when one has reached a definite end point, the dark green chloroform must be discarded and the watery layer must be extracted with fresh chloroform. It will then usually be found that the end point has not yet been reached and more methylene blue has to be added, followed by further extraction with fresh chloroform. As already mentioned, the solubility of the methylene blue dinitrophenolate in chloroform is comparatively small and it is practicable only for amounts up to about ten milligrammes of dinitrophenol to be determined by this procedure. If dealing with larger amounts, it is recommended to precipitate all of the dinitrophenol with a small excess of 0·01 N methylene blue and to determine the excess of methylene blue as described in a previous publication.[8]

As already mentioned, the test described is not specific for 2:4 dinitrophenol. A similar compound is given by 2:4:6 trinitrophenol (picric acid), and this could be differentiated from the dinitro compound by the considerably greater solubility in chloroform. In general it seems that all polynitro derivatives of phenol and naphthol with a nitro group in orthoposition to the hydroxyl group form with methylene blue an addition product which dissolves in chloroform with a green colour. Besides dinitrophenol and trinitrophenol, I had an opportunity of testing dinitroresorcinol (2:4:1:3) and dinitro-α-naphthol, which were also found to form with the dye a green chloroform solution. However, dinitro-o-cresol, the compound advocated by Dobbs and Pope, was not available for examination.

The test for the detection of dinitrophenol poisoning during the world war was an application of the so-called Derrien's test for picramic acid. This test is based on the fact that the dinitrophenol after its intake is partially reduced to amino-nitrophenol, which forms with β-naphthol a diazo dye. The technique of this test is as follows: To 10 cubic centimetres of urine add one cubic centimetre of 10% sulphuric acid and then one cubic centimetre of 0·5% sodium nitrate. The mixture is shaken and kept in the dark for five minutes. In another vessel of about 25 cubic centimetres capacity one places a freshly prepared solution of 0·5% β-naphthol in a 20% solution of ammonia. Then the diazotized urine is poured into the β-naphthol solution, mixed and allowed to stand for at least one minute. Then it is extracted with 10 cubic centimetres of ether. If after separation from the aqueous solution the ether is violet, wine coloured or orange red, the reaction is positive, that is, it indicates the presence of amino-nitrophenol. If the ether is colourless or yellow, no reaction has taken place. The result of this test is said to be always positive when applied to the urine in cases of dinitrophenol poisoning. However, as already mentioned, it depends on the presence of amino-nitrophenol, which would not be present in the gastric contents after dinitrophenol intake. It also may not occur in blood and tissues in cases of

dinitrophenol poisoning.[1] Even in urine in which dinitrophenol may still be found Derrien's test may give a negative or doubtful result. This is shown by the following example:

After the intake of two milligrammes of dinitrophenol per kilogram of body weight by the author, the urine was found to be slightly positive for dinitrophenol three hours after the intake and remained positive for the next twenty-four hours. The result of Derrien's test, however, remained negative or doubtful throughout the experiment.

Summary.

A test has been described for the detection and estimation of α-dinitrophenol, a drug recently advocated for the treatment of obesity.

Acknowledgements.

I desire to express my thanks to the Board of Directors of the Sydney Hospital for the facilities afforded to me; also to Dr. W. Keith Inglis and Dr. H. S. H. Wardlaw for their kindness while I was working in the laboratory.

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A CALIBRATION OF THE SPHERE-GAP VOLTMETERS EMPLOYED AT X RAY TREATMENT CENTRES IN SYDNEY.[1]

By WM. H. LOVE, B.Sc., Ph.D.
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SOME months ago, a calibration was made of the spark-gaps employed in conjunction with the various deep therapy plants operating in Sydney. An accurate knowledge of the potential difference on the X ray tube is of first importance in any rational scheme of X ray therapy, and unless great care is exercised the sphere-gap (spark) voltmeter gives quite unreliable results. It may be of some interest, however, to note that the sphere-gap voltmeter has been adopted as the standard method of measuring alternating potential differences by the International Electrotechnical Commission, and the way in which such an instrument is to be used has been prescribed by the British Engineering Standards Association.[1] In any circumstances, however, the sphere-gap voltmeter appears to possess certain distinct disadvantages when employed to measure the potential differences applied to an X ray tube. In the first place, it measures the peak potential just at the instant when the spark occurs, whereas the quality of the radiation emitted follows more closely the root mean square value of the potential.[2] Secondly, the sparking of the gap may set up undesirable high frequency oscillations in the tube circuit, thereby diminishing the life of the tube. From many points of view a robust form of electrostatic voltmeter is to be preferred, and it is our intention that such an instrument be built and employed in standardization work.

Calibration.

The calibration of sphere-gap voltmeters was made by the spectrometric method[3] in accordance with which the radiation from the X ray tube is spread out into a spectrum (Figure I), and the wave

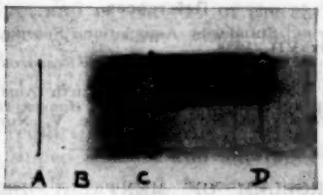

FIGURE I.
Figure showing spectrum of radiation from an X ray tube. A = undeflected beam, B = short wave length edge (λo), C = K lines of tungsten, D = K lines, second order.

length (λo) of the shortest radiation emitted is measured when the tube is operating under the potential difference to be determined. This measurement can be made with a reasonable degree of accuracy, because from Figure I it will be seen that the spectrum ends very abruptly on the short wave length side, the discontinuity serving as a very precise determination of λo.

It is known that a very simple relationship exists between the shortest wave length emitted (λo) and the maximum voltage (Vm) on the tube, the product of the two being in fact a constant, independent of all other conditions. Represented algebraically we may write:

$$Vm = \frac{12{\cdot}35}{\lambda o}$$

where λo is the minimum wave length produced, measured in Ångström units[1] and Vm is the maximum voltage on the tube in kilovolts. It will be seen that the radiation itself acts as a kilovoltmeter.

Results.

It is not proposed to give a detailed account of the results obtained, and it will be sufficient to mention the fact that in all cases the maximum potential difference as indicated by the sphere-gap

[1] This work was carried out under the control of the Cancer Research Committee of the University of Sydney and with the aid of the Cancer Research and Treatment Fund.

[1] The Ångström unit = 10^{-8} centimetres.

voltmeter was found to be distinctly greater than that shown by the X ray spectrometer. This means that the maximum potential difference under which the tube was operating was in all cases less than was believed. The percentage errors were greater in some cases than in others; but the necessary adjustments were made and it is now believed that maximum potential differences under which the tubes are operated in the various therapeutic centres are known to within something less than 5%.

Mention should be made of the fact that considerable care is required in the use of the X ray spectrometer, but a detailed description of the particular technique employed and the corrections applied cannot be given here.

This short note will serve to stress the importance of a periodic calibration of spark-gap voltmeters and, in addition to this, I would recommend that any proposed alteration in the potential difference applied to the tube should be immediately followed up with a redetermination by the spectrometric technique.

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THE PROBLEM OF MENTALLY DEFECTIVE CHILDREN IN NEW SOUTH WALES FROM THE EDUCATIONAL AND VOCATIONAL POINTS OF VIEW.[1]

By A. E. Machin, M.B. (Sydney),
Principal Medical Officer, Department of Education, New South Wales.

I wish to express my appreciation of your invitation to address you on the subject of mentally defective children.

It is my intention to bring before your notice some data and observations on mentally backward and mentally defective children in this State, and to discuss some aspects of the problems which arise in connexion with their education, training and control. I do not desire to advocate any particular course of action or definite scheme for the solution of these problems, but wish to review the position as it exists in New South Wales today, with a view, rather, that such a stocktaking may indicate what degree of urgency exists with regard to these matters and what are the possibilities of meeting the situation.

There has so far been no attempt on any large scale to deal with the problem of training and educating mentally defective children in New South Wales. As you are probably aware, the Special School at Glenfield was inaugurated in 1927 and some very valuable work has been carried out at this institution. Further reference to the work of this school will be made later.

For some years also a few special classes for mental defectives were established in several metropolitan schools, but it was possible to deal with only a comparatively small number of children.

In attempting to solve any problem of this nature it is necessary first to consider the facts of the situation. It would be necessary, for example, first of all to arrive at an estimate of the approximate percentage of mental defectives existing in the population of the State, and for our particular purpose especially, the percentage of mental defectives, educable and ineducable, of school age.

Then further questions to be considered are: What is the approximate cost of training the educable defectives? In what particular direction should they be educated and trained? What are the possibilities for successful employment in later life, and under what conditions could or should these defectives be employed?

As you are aware, a great deal of attention has been paid for a considerable number of years to the problem of training and educating mentally defective children in England and America (in fact, the first efforts in this direction in these countries date back to the middle of the last century); and a great deal of valuable experience has been gained in connexion with these matters. According to the report of the Board of Education for England and Wales for 1932, there were then 171 special schools, including residential and day schools, in England and Wales, with a total attendance roll of 16,893 children.

In this State at the present time the facilities for training mental defectives are approximately as follows: Glenfield Special School has an enrolment of 108; a few special classes exist in the metropolitan area; there is a special class for State wards at the Brush Farm Home for Girls at Eastwood; and there is a similar class for the defective boys at "May Villa", Dundas. There are also a few small private institutions and schools for defectives, and instruction is given to defectives at the Newcastle Mental Hospital. It will be seen that only the fringe of the problem has so far been touched in this State.

There still seems to be a good deal of difference of opinion, not only amongst members of the general public, but even amongst members of the medical profession, as to the desirability of incurring any great expenditure in educating and training mental defectives. This is, of course, however, largely a relative question, as it depends on the types of mental defectives that are selected for special educational treatment. At the present time the economic world is seriously dislocated and we have the unfortunate spectacle in practically every country of thousands of physically and mentally fit members of the community being unable to obtain employment; and, as far as can

[1] Read at a meeting of the Section of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association on November 21, 1933.

be judged at the present time, thousands will continue to be unemployed for a considerable period unless some new methods of economic adjustment are found to solve the problem.

Ignoring this aspect of the general economic situation for the time being, however, one might consider the advantages, possibilities and end results of a scheme of education and training for mental defectives.

It has been the experience in England and America, I understand, that only a certain proportion of educable defectives can be successfully absorbed and employed in the outside world, and even for these after-care and vocational committees are necessary to assist them in obtaining employment and generally to supervise the circumstances of their existence. Without further tutelage and supervision the final possibilities with regard to mental defectives who have gone through a course of training in a special school or vocational establishment are limited and depend on different factors. In many cases it would be possible for the defectives to be suitably employed by their own families, for example, where the families follow agricultural pursuits or trades or unskilled occupations in which the defectives could be usefully employed.

It is obvious that in the case of all mental defectives semi-skilled and unskilled manual occupations afford the only field of opportunity for employment, and amongst these agricultural pursuits present the greatest possibilities. For girls, of course, the chief avenues are those of general domestic work, laundry and sewing. The employment of subnormal girls, other than in their own homes, however, presents a difficult problem.

Then again a large group of employable children could be later employed only in institutions, such as farm and industrial colonies, where they would be under continual supervision and protection. Unless these types of institutions are available for this class of defective, the training of such defectives would be largely wasted.

It is evident that the problem of dealing with defectives is a very wide one, and for its full solution will require cooperation on the part of different sections of the community, the State being, of course, chiefly involved both in organization and expenditure.

There are, however, other aspects to be considered besides those of the economic possibilities. The mental defective who has gone through a suitable course of training is in every way a more satisfactory member of the community. Even if he returns to his home the general improvement in his habits and morale is a great asset, not only to himself, but to other members of the family; it makes life for all much more comfortable and satisfactory. This improvement in habits and character is of especial value to those individuals who are liable to make more or less uncontrolled and unprotected contact with the outside world. Self-control, self-reliance and morale are in the majority of cases increased by proper training.

In this respect one must except the defectives with vicious and criminal tendencies who, as experience has shown, use their increased knowledge and capacity as a rule only for carrying out their vicious acts or habits in a more cunning manner. According to English data, however, only a very few are of the inherently amoral and incurable types.

Then again we have to take into consideration the humanitarian aspect of the question of educating and training mental defectives, as there is no doubt that their happiness is greatly increased, in most cases, by the encouragement which is given them to develop their limited capacity, and by their efforts to become useful members of society.

The education of mental defectives, however, presents a very difficult problem. If they attend the ordinary school or class, there may be undesirable repercussions from inability to make headway under these conditions, with possible serious effects on the conduct and psychology of the child, leading in some cases to truancy and delinquency, as pointed out by Dr. Bruce in connexion with the work at the Children's Court. On the other hand, their education in special schools is much more expensive, requiring specially trained teachers, specially equipped schools and smaller classes.

Unless specially trained, however, or able to be employed in the circumstances previously mentioned, the defectives find, as adults, that they are more or less useless members of the community and remain a burden either on the State or on their family, or on both. There is no need to enlarge on the train of social evils which may follow as a result of lack of proper care, control, or means of support of defectives, as these are well known to most.

The Department of Education of New South Wales, through the School Medical Service, has for many years carried out a considerable amount of mental testing and psychological examination of mentally backward and defective children. This work has been carried out in the schools, at the Children's Court, and in connexion with the medical examinations carried out for the Child Welfare Department. In addition, medical officers of this service, acting in an honorary capacity, have mentally assessed and classified all cases coming before the Society for Crippled Children. Since the inauguration of the society in 1929 some 1,200 children and adolescents have been examined.

During the years 1925-1926-1927 a survey of mental defectives in attendance at primary schools throughout New South Wales was carried out by school medical officers under the direction of Dr. (now Professor) Harvey Sutton. The findings in a representative group of schools with a total enrolment of 76,557 children are shown in Table I.

It will be noted that the percentage of defectives was found to be 0·88, 0·35 being medium grade and 0·53 high grade and borderline cases. It is con-

sidered, for various reasons, that this figure is probably below the real incidence. As can be readily understood, some cases would be missed.

Table I.

Findings from survey of 39,104 boys and 37,453 girls, totalling 76,557 children.

Age Group.	Sex.	Mentally Defective Group. A. Low Grade	B. Definite, but not Low Grade.	C. Borderline, High Grade.
Under 6 ...	M	—	1	1
	F	—	1	—
6-14	M	3	133	222
	F	8	104	177
Over 14 ...	M	—	7	6
	F	1	11	2
Totals ...	M	3	141	229
	F	9	116	179
Percentages mentally defective		—	0·35	0·53

Since 1927 school medical officers have continued to test and examine mentally backward children at all schools visited, but the tests are limited to children under twelve years of age not previously tested, and selected by the teachers on the grounds of being two years or more scholastically retarded. It has not been possible, therefore, to collate further statistics with regard to the incidence of mental deficiency. I would mention, incidentally, that teachers are requested to enter on the mental survey schedule and submit for examination any pupils, whether backward or not, showing marked nervous or psychological abnormality.

Including the borderline cases (that is, those with an intelligence quotient of 70 to 75), it is considered that approximately 1% represents the average incidence of defectives in attendance at schools in this State. As the total enrolment of primary schools in the State is approximately 400,000, it is calculated that the defectives of school age number about 4,000.

According to information supplied to the Mental Deficiency Subcommittee of the Council of Mental Hygiene, through the courtesy of Dr. Hogg, Inspector-General of Mental Hospitals, there are 309 mental defectives under the age of sixteen in the mental hospitals.

Definition and Diagnosis of Mental Deficiency.

These observations bring us to the important question of the definition and diagnosis of mental deficiency.

The definition of feeble-mindedness, that is, the upper level of mental deficiency, given in the British Act, is as follows:

> Feeble-minded persons—that is to say, persons in whose case there exists mental defectiveness which, though not amounting to imbecility, is yet so pronounced that they require care, supervision, and control for their own protection, or for the protection of others, or in the case of children, that they appear to be permanently incapable by reason of such defectiveness of receiving proper benefit from the instruction in ordinary schools.

Such a definition allows for considerable latitude in interpretation.

Definitions are also given for imbecility and idiocy, which are now also included under the term "mental deficiency".

In estimating the degree of intelligence in this State, the Binet tests, as adapted for Australian children by Dr. Phillips, have been applied. Although the intelligence quotient is a valuable and necessary guide in estimating the general ability or capacity of the child and so in classifying him, it is by no means the only factor to be taken into consideration. It is the mind and personality, not only the intellect, which have to be investigated. The intelligence quotient is a fairly reliable test of the scholastic educability.

The capacity of the defective child for useful employment depends almost as much on his emotional stability and powers of social adaptability as on his intelligence quotient. These qualities have to be judged, as no reliable tests for quick estimation have been evolved. A period of observation, under satisfactory conditions of environment, is necessary in some cases to the formation of a correct opinion. The emotionally stable and sensible type of defective possesses in fair measure the faculty (as it has been well put) of "properly regulating his conduct in the more important affairs of his life", and so is more likely to succeed occupationally.

On the other hand, even the high grade or borderline defective with a relatively high intelligence quotient may fail because of emotional instability and poor capacity to adapt himself to his environment.

In the school medical work we have taken as a working standard an intelligence quotient of 70 to 75 as indicating a possibility or suspicion of feeble-mindedness, and an intelligence quotient of 70 as the upper limit of the probably or definitely feeble-minded.

The chief requirement for school purposes is, however, the classification "S.S.S.", that is, suitable for a special school or class and unsuitable for instruction in the ordinary school or class. Even though at the present time there are only very limited facilities available for special training of these children, the results of the testing and examination are of distinct value to the teacher.

It will be observed, by referring to Table I, that comparatively few low grade (below intelligence quotient 50) defectives find their way into the schools (and actually fewer of these exist) and the ratio of the higher grades to the medium grades is as 0·53 to 0·35. In fact, if we take the intelligence quotient of all children, we find that the curve of incidence of mentally backward children rises progressively with the intelligence quotient. This is, of course, what one would expect.

It will be seen, therefore, that the problem, from the school point of view, is not only that of educating the feeble-minded, but also the large number of backward children who are difficult to

teach in the ordinary class. However, I do not wish to discuss this aspect of the problem, except in so far as to say that some of these children would be better educated in a special school or class, and that the remainder could be provided for in the ordinary school by the adoption of special methods of class grading. Because of their larger numbers, as far as the teacher is concerned, they do present a special educational problem, and, as can easily be imagined, the percentage of mentally defective and backward children varies very much in different schools and different districts.

It has not been found possible, in connexion with the school work, to obtain reliable data and to prepare statistics on such issues as causation factors and other points of interest from the medical point of view. It is especially difficult to get information with regard to circumstances of heredity, as parents are as a rule either reticent or ignorant concerning their family histories. The large amount of experience gained in examining and testing mentally defective children in the schools has, however, made it possible to make general observations and arrive at certain general conclusions. It has been observed, for example, that primary amentia is in a great many cases obviously due to hereditary influences, and it would appear that some of the modern social benefits tend to encourage the propagation and survival of primary aments in the community.

One frequently meets with a family at school in which all the children range from very dull to mentally defective. When information is available in these cases, it is always possible to confirm unsatisfactory mental or nervous conditions in one or both parents. It may be found that one or both are of inferior mentality, defective, unstable, or intemperate, or give a personal or family psychopathic history.

An example that I will cite is as follows:

The father is emotionally unstable, highly excitable, bad tempered, and given to physical violence towards people for slight grievances; the mother is of a poor mental type; three children attending school are all low grade defectives.

In other cases, of course, no such influences are evident, and frequently no facts or history whatever which would account for the occurrence of the amentia can be adduced.

It has been observed also that a very large proportion of the medium and high grade defectives who are in attendance at school present no marked physical defects or disabilities. In fact, one is impressed over and over by the very good physique of many of these children. This is an important aspect of the matter, as they would be necessarily confined to a manual occupation. Also only a very small number present obvious or certain signs of luetic infection, although, of course, this has been considered to be one of the causes of mental deficiency. In regard to nineteen State wards in attendance at the Glenfield Special School, Dr. Cooper Booth found that the Wassermann test in all failed to yield a reaction. This is admittedly only a very small number of cases, but one would have expected it to be likely soil for such a test.

With regard to causation factors in mental deficiency, it has been possible to obtain some data in a series of crippled and orthopædic cases. The tabulated results of an analysis in 1,029 cases (children and adolescents) clinically examined and mentally assessed at the clinics arranged by the Society for Crippled Children are shown in Tables II and III.

In Table II the total number of cases is 1,001. The total mentally defective is 144, or 14·4%. The 28 non-orthopædic cases of congenital imbecility and idiocy have not been included in the totals.

In Table III the total mentally defective is 144. The percentage mentally defective is 14·4%. Of the 144 defectives, 100, or 69·4%, suffered from spastic paralysis.

The clinical examinations were carried out by orthopædic surgeons, and the mental tests by officers of the School Medical Service. The children submitted are not all "cripples" in the ordinary sense of the word, but include those with various types of orthopædic conditions and twenty-eight children with non-orthopædic congenital imbecility

TABLE II.
Society for Crippled Children Cases.

Condition.	Description.	Has Attended or is Attending School.	Schooling Nil or Negligible.	Under School Age.	Total.
Spastic paralysis ..	Average	40	8	20	68
	Backward	19	7	8	34
	Mentally deficient	16	69	15	100
Infantile paralysis .	Average	244	10	75	329
	Backward	34	4	6	44
	Mentally deficient	4	5	—	9
Congenital deformities	Average	60	3	44	107
	Backward	7	5	3	15
	Mentally deficient	2	2	3	7
Congenital imbecility and idiocy—					
Non-orthopædic .		5	10	13	28
Orthopædic		—	3	5	8
Miscellaneous	Average	155	23	37	215
	Backward	32	10	3	45
	Mentally deficient	5	11	4	20

and idiocy. We meet here chiefly the developmental and acquired or accidental types of amentia. In some cases an original primary type of amentia may have been present.

TABLE III.

Physical Defect.	Percentage of Cases (nearest figure).	Percentage Mentally Defective.
202 Spastic paralysis	20·2	49·5
382 Infantile paralysis	38·2	2·3
417 Miscellaneous	41·7	8·3

Total 1,001 cases (not including 28 non-orthopædic cases of congenital imbecility or idiocy).

The outstanding facts which these tables reveal with regard to this series of cases are as follows: (i) That the most important cause numerically of partial physical disability or crippling is infantile paralysis, but that patients with this disease show only a slight increase in incidence of mental deficiency, namely, 2·3%. (ii) That both the highest incidence and the greatest number of cases of mental deficiency occur in the spastic group. Of the average incidence of 14·4% of mental deficiency, 9·9% was accounted for by cases of spastic paralysis. (iii) That 49·5% of the patients with spastic paralysis were found to be mentally defective, it being apparent that, in the event of disease or injury of the Rolandic areas, the intelligence centres are very liable to be involved.

Also to be noted is the large number of spastic children who failed to attend school, 84 out of 202 children of school age or over having never attended school, or only for a year or so. Both the crippling condition and the deficiency would be responsible for non-school attendance, either together or separately in different cases.

I would mention here that mentally defective children are not excluded from attendance at public schools, except on the grounds of unsatisfactory conduct, or a very low grade of amentia. Children, whether backward or not, are excluded if they take epileptic fits during school hours, as, amongst other reasons, it is considered that the effect on the other children is undesirable. A special school or institution for epileptics is desirable, but none exists so far. If educable, epileptics can receive tuition through the correspondence school.

The unsatisfactory conduct of mental defectives may be of such a nature as to affect the discipline of the class, to affect other children adversely, or to be a source of danger to themselves. Some are not amenable to school discipline or control, others are given to viciousness or violence, and some, as puberty is reached, exhibit precocious sexual propensities which necessitate their exclusion from school attendance. In the majority of cases, however, conduct is fairly satisfactory.

As the teacher of the ordinary class has very little time to give individual attention to each child, in cases below a certain standard of backwardness, very little, if any, progress in education results from attendance at school; and children so affected often are automatically promoted merely on account of age and size.

Some of the higher grade defectives and borderline children do, however, succeed in making some progress through the efforts of enthusiastic and hard-working teachers, who manage to find some spare time to give them individual attention, but even in these cases the training in the ordinary school does not meet their particular requirement, which is rather to be fitted in some way for employment in adult life.

The attendance of the defective child at the ordinary school may have serious psychological repercussions. Discouragement and disinterest, which naturally follow as a result of inability even to make any useful effort towards educational advancement, lead in some cases to truancy and delinquency.

As I have stated previously, it may be possible for the defective, on leaving school, to be more or less usefully employed by his own family; in other cases the financial circumstances may enable him to remain at home indefinitely; some develop criminal tendencies and very many become in one way or another a responsibility on the State.

Special Schools and Classes.

I wish now to refer to the experience gained in connexion with the Glenfield Special School and with a few special classes for mental defectives which were established in the ordinary schools. Some years ago several special classes for feeble-minded children were formed in the metropolitan area. The results of this experiment were not so satisfactory as anticipated, but they represented a definite attempt to meet the problem in the interest of the child.

First of all there is the difficulty of the environment of the ordinary school, no separate playground being available, and so on. Some conspicuousness attaches to the fact that the children are relegated to the special class. Then the pupils of such a class are drawn from a fairly limited area and both their mental and chronological ages vary tremendously. Also it is not possible to make the most satisfactory arrangements for training in manual work.

The opinion is therefore held that the institution alone of special classes would not result in a satisfactory solution of the problem of training feeble-minded children. Special classes should, however, admirably meet the problem of educating in the ordinary schools the dull and backward children (though not feeble-minded) and might serve to initiate a scheme of education for defectives, remaining permanently in areas or schools where the above-mentioned drawbacks could be obviated.

However, for satisfactory results to be obtained, it is considered that the education and training of the feeble-minded need to be carried out in special

schools or institutions, staffed with suitably trained teachers and provided with the necessary equipment for manual training. Some of these schools could be day schools, but some residential schools would be necessary, for example, to give training in agricultural pursuits and to cater for country children *et cetera*.

The Glenfield Special School was established and commenced operations in 1927, the present enrolment being 108. All the pupils are in residence at the school. Medical supervision of the children is maintained by the School Medical Service.

I wish to acknowledge my indebtedness to Dr. Phillips, principal of the school, for data and information which he has supplied.

Experience in connexion with this school has shown that the desiderata and contraindications with regard to suitability for training in a special school are as follows: The intelligence quotient should be within the range of 55 to 75 approximately. Depending on behaviour, stability and environment, some children with an intelligence quotient even up to 80 would be better in a special school. It is probable that 60 represents the lower limit desirable in the great majority of cases. The minimum mental age should be five. The most suitable chronological age at admission would, therefore, be about eight to ten years, but children over the age of twelve are considered to be too old for admission to a special school.

Children who give a history of delinquency are not refused, but if delinquency is exhibited after admission to the school, they are excluded. There has been very little trouble in this respect. Physical handicap renders some unsuitable for tuition or training in a special school.

It will be noted that the upper level of the intelligence quotient for a special school is given as 75. It is considered that the majority of mentally backward children with an intelligence quotient above this level should be able to be dealt with in some way in the ordinary school.

A brief outline of the objects and methods of the teaching and training at the Glenfield Special School should be of interest. Children are trained in correct habits and conduct. Only limited scholastic attainments are aimed at, that is, they are taught to read a newspaper, write a letter, and calculate change and the ordinary weights and measures.

With regard to this aspect of their education, the results that can be obtained are surprising. Many are able to read as well as pupils of the fifth class standard, but they find more difficulty with figures and calculations.

Dr. Phillips has kindly furnished me with some specimens of the work (unaided composition and letters), the intelligence quotient of the child having been noted on each specimen. I think you will agree that the standard which the children reach in these matters is higher than one would expect. We realize that some hold that it is not worth while devoting much time to the teaching of the "three R's" in these cases, but an examination of the work of these children will show that much can be accomplished in this direction.

The pupils are taught manual work, such as rug and mat making, basket work and raffia work, wood work *et cetera*, not only with the object of teaching them simple productive occupations, but for the important purpose of developing eye and muscle coordination and control. In many of these children muscle control and coordination are very poor and some are afflicted with athetoid movements.

Dr. Phillips has found that the wheeling of a wheelbarrow is an excellent exercise for these extreme cases.

He relates one instance in which the boy's gait was so unsteady and his athetoid movements were so marked that he was unable to wheel a barrow without losing the load after a very short distance. However, with perseverance, he gradually improved, and finally was not only able to transport material successfully in the barrow, but was able to gather in the eggs without any breakages.

I merely quote this as an instance of the improvement which can be obtained in these cases by proper training.

The school has quite a considerable area of land, and the boys are taught agricultural pursuits. They learn to handle animals very well, to plough and generally to carry out farming work quite efficiently.

The girls are trained in general domestic work and the senior girls do cooking and make their own dresses.

Sport and athletics form a part of their recreation activities, as in ordinary schools, and quite commendable skill is shown by many. The sporting activities have provided an instance of the importance of the factor of emotional stability and innate social capacity. The boy who has shown the greatest ability as organizer and captain in sport has an intelligence quotient of only 54, with correspondingly poor scholastic attainments.

The results generally of the course of training at the school have been very encouraging, but attendance for at least four to six years, and until sixteen years of age, is essential for a satisfactory grounding.

In considering the question of educating mental defectives, an important aspect is that of possible mental deterioration or improvement. Table IV shows the results of a series of mental tests carried out on 92 defectives at Glenfield over an average period of four years.

In this group the intelligence quotient remained stationary in 58, decreased at least 10% in 12, and 10 points in 14, and increased at least 10% in 4, and 10 points in 4. Therefore, 15·2% showed definite deterioration and 13% showed slight deterioration.

In one instance improvement in the intelligence quotient took place up to the age of eighteen years. As it is now considered that full intellectual

Table IV.

Results of revisionary mental tests on 92 children at Glenfield Special School over an average period of four years.

Group.	Intelligence Quotient Increase.		Intelligence Quotient Stationary.	Intelligence Quotient Decrease.	
	10% or More of Original Intelligence Quotient.	10 Points or More.		10% or More of Original Intelligence Quotient.	10 Points or More.
Boys	4	4	33	8	9
Girls	—	—	25	4	5
Total	4	4	58	12	14

15.2% show definite deterioration, 13% show slight deterioration.

development is reached at about sixteen years of age (though further investigation of this question is necessary), such a case appears to indicate delayed or retarded development.

A marked improvement is usually found in cases of partial deafness, which disability has increased the apparent backwardness.

It has been observed that the low grade defectives tend to deteriorate mentally, as do also children who suffer from epilepsy and *encephalitis lethargica*. Special training of the lower grade feeble-minded would appear, therefore, taking all things into consideration, not to be justified.

Dr. Phillips tries to keep in touch with the ex-pupils of the school. He has ascertained that of those who have remained at the school until fifteen years of age, over 60% are in employment. As would be expected, some lose their positions for various reasons. It is obvious, however, that if defectives were trained on any large scale, grading according to capacity for employment under different sets of conditions would be necessary, together with opportunities for varying degrees of supervision through after-care and welfare organizations for those who are employed amongst the general population.

For the proper operation of any comprehensive scheme instituted for the education and training of mental defectives, certain legislation (similar, for example, to that which has been enacted in England —*Mental Deficiency Act*, 1913, *et cetera*), which would provide for the detection, education and proper care and control of mental defectives, would be necessary.

Perhaps more necessary than the provision of facilities for the special education and training of mental defectives is the establishment of special institutions, such as industrial and farm institutions or colonies, and special institutions chiefly for the care of mental defectives.

In fact, for certain types of children the sojourn in the special school would serve rather as a test of their suitability for special training or admission to an institution for the care of defectives.

Then again, legislation is necessary to deal effectively with the unstable and vicious type of defective. These children are not suitable for a special school and require to be admitted to an institution, intermediate in type, between a mental hospital and a reformatory.

From experience gained so far, one would be inclined to say that the medium and high grade defectives only are suitable for education in a special school, but that this training is not justified on the results which are likely to be obtained in the case of many of the medium grade feeble-minded and most of the low grades, that is, those with an intelligence quotient of 55 and under.

Summary of Facts and Conclusions.

It would appear that there are somewhere about 4,000 mentally defective children of school age in New South Wales, many of whom require education in special schools or classes and who cannot be satisfactorily educated in the ordinary school or class.

To meet fully the problem of educating and training mental defectives of school age in this State, the establishment of day schools and additional residential schools, and possibly special classes in the ordinary schools, would be necessary. In contemplating any efforts in these directions, however, the cost would have to be taken into consideration.

As a corollary to such a scheme of education, it would be necessary further to establish various types of institutions or colonies for the training, care, and/or employment of mental defectives of all ages. To enable such activities to be carried out, however, the enactment of certain legislation with regard to mental defectives would be necessary.

Acknowledgements.

I wish to acknowledge my indebtedness to Mr. G. R. Thomas, B.A., Under-Secretary and Director of Education, New South Wales, for permission to use departmental information and data, and to the New South Wales Society for Crippled Children for their courtesy in giving access to the records of their cases.

Reports of Cases.

SUPPRESSION OF URINE AS A TERMINAL COMPLICATION OF CHRONIC NEPHRITIS WITHOUT CALCULUS.

By A. R. Buchanan, M.D. (Melbourne), M.R.C.P. (London),
Medical Clinical Assistant, Alfred Hospital, Melbourne.

Suppression of urine is not a common condition, and as a complication of chronic nephritis without calculus it is sufficiently rare to merit attention. In view of this, the following cases are of unusual interest.

Case I.

R.F., a male, aged fifty-nine years, was admitted to hospital on September 13, 1932. No history of any previous illness was obtained. The patient stated that he had been quite well until a few weeks before his admission, when he became more easily tired and rather listless. This gradually became worse until twelve days before entering hospital, when he had to take to his bed. At that time he noticed that he was passing very little urine.

On admission to hospital the patient was very drowsy and the history was difficult to obtain. He was a pale man who looked his years. The tongue was dry and covered with a brown fur. The heart was enlarged, the apex beat being located in the fifth intercostal space, 11·25 centimetres (four and a half inches) from the mid-line. The cardiac sounds were clear, but of poor quality; the cardiac rhythm was regular. The systolic blood pressure was 170 millimetres of mercury, the diastolic pressure 120 millimetres.

No œdema of the subcutaneous tissues was noted. Examination of the *fundus oculi* revealed the presence of retinal arteriosclerosis. A radiographic examination of the urinary tract failed to reveal any evidence of calculus.

While in hospital he passed no urine and gradually became worse despite treatment; his drowsiness and mental clouding deepened insensibly into coma, in which he died. The urinary bladder was not at any time palpable above the pubis. He died on September 18, 1932.

Post Mortem Findings.

Necropsy was performed on September 19, 1932. The salient features were as follows:

The heart was found to be somewhat enlarged, owing mainly to hypertrophy of the left ventricle. The cardiac valves were normal. The coronary arteries showed slight atheroma. There was a moderate degree of atheroma in the aorta.

The kidneys were enlarged, together weighing 453 grammes (sixteen ounces). The capsule of each kidney was thickened and adherent, peeling with difficulty, and leaving a mottled red finely granular surface consisting of very slightly raised greyish granules with a sunken red reticulum. There was slight reduction in the depth of the cortex, with general distortion of the cortical pattern and a blurring of the line of demarcation between the cortex and the medulla. There was moderate prominence of the arcuate arteries, and some increase in the amount of pelvic fat. There was no dilatation of the pelvis of either kidney, nor was there any dilatation or obstruction of either ureter or of the urethra. The urinary bladder appeared normal and contained about five cubic centimetres of clear urine.

The other organs showed nothing noteworthy.

Microscopic sections of the kidneys revealed general fibrosis with marked thickening of the blood vessels. The glomeruli showed all stages of degeneration, through the stage of adherence to Bowman's capsule up to the stage of complete atrophy and replacement by a mass of fibrous tissue; a few small retention cysts were present. Very marked round cell infiltration was everywhere seen, but particularly around the cortical and convoluted tubule zone.

Case II.

H.H., a male, aged thirty-six years, was admitted to hospital on November 11, 1930. He stated that he had passed no urine for the past forty-eight hours, but apart from slight headache felt well.

He had suffered from scarlet fever at the age of six years; otherwise he had been quite well. There was no history of nocturia or other untoward symptoms.

On admission to hospital the patient was a pale, thin man who looked older than his years. The tongue was dry and covered with a brownish white fur. The heart was enlarged, the apex beat being located in the fifth intercostal space, 13·75 centimetres (five and a half inches) from the mid-line. The second sound at the aortic base was accentuated and ringing; the other cardiac sounds appeared clear; the cardiac rhythm was regular. The systolic blood pressure was 178 millimetres of mercury, the diastolic pressure was 120 millimetres. The peripheral arteries were thickened and rather tortuous. The urinary bladder was not palpable above the pubis. No œdema of the subcutaneous tissues was noted. Examination of the *fundus oculi* revealed the presence of advanced renal retinitis with much exudate, many small hæmorrhages and a moderate degree of papilloedema.

A radiographic examination of the urinary tract failed to reveal any evidence of calculus.

While in hospital he passed no urine, and gradually became worse despite treatment, but in contrast to the previous patient, the mentality remained quite clear and he was able to cooperate well and describe his symptoms, which were very distressing. Headache became prominent and was soon followed by intractable vomiting. Shortly before death a sudden amaurosis developed. He died on November 14, 1930.

Post Mortem Findings.

Necropsy was performed on November 15, 1930. The salient features were as follow:

The heart was found to be somewhat enlarged, owing mainly to hypertrophy of the left ventricle. The myocardium showed no gross change. The cardiac valves were normal. There was some slight atheroma of the coronary arteries and moderate atheroma of the aorta.

The kidneys were of normal size and together weighed 312 grammes (eleven ounces). The capsule of each kidney was thickened, but stripped easily from a pale mottled and pitted subcapsular surface. There was marked reduction in the depth of a pale, streaky cortex, with general distortion of the cortical pattern and a blurring of the line of demarcation between the cortex and the medulla. There was moderate prominence of the arcuate arteries and some increase in the amount of intrapelvic fat. There was no dilatation of the pelvis of either kidney, nor was there any dilatation or obstruction of either ureter or of the urethra.

The urinary bladder showed no gross change and contained only about five cubic centimetres of clear urine.

The other organs showed nothing noteworthy.

Microscopic sections of the kidneys revealed moderate generalized interstitial fibrosis, which was more marked in the glomerular region. The glomeruli, though the number involved was not as great as in the other cases, showed gross pathological change of all degrees up to complete atrophy and replacement by fibrous tissue. There was a marked engorgement of the kidney and a considerable round cell infiltration of the cortical zone.

Case III.

M.S., a female, aged sixty-three years, was admitted to hospital on April 27, 1927. She stated that she had been ill for some months with pain and swelling of the small joints of the hands. She further stated that she used to pass a large quantity of urine and had suffered from nocturnal frequency of micturition, but recently she had been passing much less urine. In the twenty-four hours immediately prior to admission she had suffered from marked diarrhœa, but during that time she had passed no urine. No history of any previous illness was obtained.

On admission to hospital she was a stout, rather pale woman, with deformity and swelling of the small joints of the fingers. The tongue was dry and covered with a brown fur.

The heart was enlarged, the apex beat was located in the fifth interspace, 10·8 centimetres (four and a quarter inches) from the mid-line. The cardiac sounds were clear, but "tic-tac" in type. The cardiac rhythm was regular. The systolic blood pressure was 165 millimetres of mercury, the diastolic 110 millimetres.

Examination of the *fundus oculi* revealed the presence of retinal arteriosclerosis.

A radiographic examination of the urinary tract failed to reveal any evidence of calculus. There was no œdema of the subcutaneous tissues.

While in hospital she voided no urine, but diarrhœa was a prominent symptom. Her condition became gradually worse and she died on May 1, 1927.

Post Mortem Findings.

Necropsy was performed on May 2, 1927. The salient features were as follows:

The heart was enlarged, owing mainly to some hypertrophy of the left ventricle. The myocardium showed no gross change. The cardiac valves were normal. There was slight atheroma of the coronary arteries and moderate atheroma of the aorta.

The kidneys were of normal size and together weighed 326 grammes (eleven and a half ounces). The capsule of each kidney was thickened and slightly adherent to a deeply congested, smooth subcapsular surface, which showed ill-defined opaque white areas in a red background. There was only slight blurring of the cortical pattern, but there was very marked streakiness of the cortex and a slight blurring of the line of demarcation between the cortex and the medulla. There was increased prominence of the arcuate arteries. There was no dilatation of the pelvis of either kidney, nor was there any dilatation or obstruction of either ureter or of the urethra. The urinary bladder showed no obvious abnormality, and contained only about five cubic centimetres of clear urine. The other organs showed nothing noteworthy.

Microscopic sections of the kidneys revealed an advanced generalized fibrosis with thickening of the blood vessels. The glomerular changes were extremely advanced and hardly a whole glomerulus was to be seen. The round cell infiltration was intense, much more marked than in the other cases; a possible reason for this may be the toxæmia responsible for the joint change.

Discussion.

In each of the three cases reported here death occurred five days after the onset of suppression of urine. This fact is worthy of note in as much as the average duration of life in cases of suppression due to unrelieved obstruction, for example, "calculous anuria" is nearly twice this time. The writer suggests as a possible explanation the following sequence of events. Owing to the presence of gross renal damage before the onset of suppression the duration of the "latent period or period of tolerance" is extremely brief; during this time in a less damaged kidney the toxic products of renal failure, whatever they may be, mount to a concentration high enough to cause symptoms. In the cases here recorded the second period, namely, the "period of intoxication", becomes apparent with the onset of suppression, as the patient has existed on the verge of uræmia for some time. This fact is well illustrated in the history given in the first case, though not so clearly indicated in the other two.

Microscopically the kidneys in the three cases resemble one another very closely, although the underlying ætiology is undoubtedly different. All show an advanced condition of interstitial fibrosis with glomerular change, advanced in Cases I and III, less advanced in Case II, while with this fibrosis is associated a round cell infiltration, the intensity of which runs parallel to the degree of sclerosis.

It is very interesting to note that the round cell infiltration in Cases I and III, in which it would appear that the renal changes are primarily arteriosclerotic or degenerative in origin, is much more intense than in Case II, in which it is possible that a past acute scarlatinal nephritis is the basis of the pathological change.

It is to be regretted, as these cases all occurred in an infirmary, that the clinical notes are brief and that it was impossible to attempt any biochemical investigations. Such investigations would doubtless have enhanced the value of this note.

Summary.

1. Three cases in which suppression of urine has occurred as a terminal complication of chronic nephritis are presented.

2. In each case death occurred five days after the onset of suppression.

3. The symptoms seen in these cases, namely, increasing coma, headache, amaurosis, vomiting and diarrhœa, are those usually seen in uræmia.

4. The microscopical picture presented by the kidneys in each case is in essentials the same.

Acknowledgements.

In conclusion, I wish to express my grateful thanks to Dr. J. A. Braxton Hicks for his assistance in the correlation of the microscopic pathology, and to Dr. A. P. Piggot for his help in obtaining certain of the clinical and autopsy notes.

Reviews.

PÆDIATRICS.

That pædiatrics is actually an extension of general medicine continues to be the main theme in this, the fifth edition of Dr. John Thomson's "Clinical Study and Treatment of Sick Children", edited by Dr. Leonard Findlay; and the book remains limited to the consideration both of special varieties of morbid processes as modified both in course and appearance in the growing patient, and also of the methods of examination applicable to the very young. It is intended as a guide to the general practitioner and the advanced student, and may be deemed an "appendix" to works on general medicine.

The chapters on nutritional disease, rickets, spasmophilia and the "acid intoxications" have been entirely rewritten. With tuberculosis, the significance of the cutaneous tests, the relative frequency (in Great Britain) of the human and bovine types and the clinical incidence of the disease in its various forms have been thoroughly discussed.

In chorea and rheumatic fever a very welcome emphasis is laid on the necessity for the follow-up treatment of these types of rheumatic infection. There is a good description of the methods of performing "cistern" and "ventricular" puncture. The chapter on the everyday care of the deaf and blind and of mental and cardiac cripples is again a feature of the book. Mention is made of many of the newest methods of diagnosis and treatment, usually with a definite opinion as to their value or otherwise without long discussion. There are numerous and excellent illustrations, a chapter on hygiene and therapeutics and an appendix including diet lists, prescriptions *et cetera*.

[1] "The Clinical Study and Treatment of Sick Children", by J. Thomson, M.D., LL.D., F.R.C.P.; Fifth Edition, rewritten and enlarged by L. Findlay, M.D., D.Sc., M.R.C.P., F.R.F.P.S.G.; 1933. Edinburgh: Oliver and Boyd. Demy 8vo., pp. 1109, with 344 illustrations. Price: 30s. net.

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RAMISECTION AND SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM.

In October, 1930, attention was drawn in this journal to a report that appeared in *The Lancet* on the value of ramisection in spastic paralysis. The report was signed by well known surgeons and others in England, who concluded that the operation of cervical or sympathetic ramisection as performed by Dr. N. D. Royle was without effect upon the rigidity from extrapyramidal disease. They held that the operation appeared to have no place in the treatment of spastic weakness. In our discussion on the subject reference was made to the claims of certain Australian and American surgeons who held that the operation did afford relief to sufferers from spastic paralysis. The suggestion was made that a special committee in Australia should take the matter up and inquire into the condition of patients both before and after operation over a period of two or three years. The Royal Australasian College of Surgeons was named as the body best suited for such an investigation. The suggestion was adopted and a committee in Melbourne undertook the investigation. The report of that committee has come to hand and is published in this issue.

The Committee's main conclusion is that sympathetic ramisection is of no permanent value in the treatment of spastic paralysis—a conclusion that agrees with that of the London investigators, though it is not so strongly worded as was their report. This conclusion must be accepted. In accepting it, we must draw attention to two facts. The first is that nearly twelve months ago Dr. Royle abandoned the view that the effects of ramisection claimed by him were due to the division of sympathetic nerves supplying voluntary and involuntary muscles. This recantation was made necessary by the work of Wilkinson and others, who showed that unmedullated nerves from the sympathetic rami do not supply voluntary muscles. In a paper published in this journal on May 6, 1933, Dr. Royle's conclusions were as follows:

1. The results of sympathetic ramisection are explained as due to alteration in the circulation of the spinal centres.

2. The effects of sympathetic thoracic trunk section are due to the alteration of circulation of pre-spinal centres, particularly the vasomotor centre.

3. The sympathetic does not directly innervate the viscera, but effects alteration in function through alteration in blood supply. This applies to both abdominal and thoracic organs.

The second fact is that in place of ramisection Dr. Royle has substituted an operation of thoracic trunk section. That this alteration in the attack on the sympathetic nervous system will materially affect the results obtained in the treatment of spastic paralysis is unlikely and remains to be proved. Admittedly division of the sympathetic trunks is not without effect on muscles or viscera. If Dr. Royle's view of the effect on the circulation be correct, it remains to be seen whether the effect is permanent. Further, even if the circulation in muscles and viscera is affected, we do not know that no other effects are produced. The mystery of the sympathetic nervous system is not yet unravelled.

It is not right to refer to the function of the sympathetic nervous system, to discuss sympathetic ramisection and sympathetic trunk section and to go no further. Great advances have been made in the surgery of the sympathetic and these advances have been very largely the result of the work of Dr. Royle and of his early collaborator, the late John Irvine Hunter. Those who have followed

Dr. Royle's investigations know that he has worked consistently in the experimental laboratory as well as on the human body—he made the laboratory his approach to the operating theatre. The world of surgery is in his debt. At the recent congress in Hobart he reported results on the brain of operations on the sympathetic nervous system and claimed that the effect produced on the circulation in chronic hydrocephalus was beneficial. He also claims to have produced changes in the pancreas that have altered the clinical course of *diabetes mellitus*. This work will doubtless be investigated and controlled by other workers. Work of this kind may prove to be extremely valuable and will be likely to play a large part in solving the riddle of the sympathetic nervous system.

Current Comment.

THE THERAPEUTIC VALUE OF PSYCHOANALYSIS.

THE term psychoanalysis, like many another in popular use, is widely misunderstood. There is a prevalent interest in the whole subject of psychology, and in the realm of therapeutics any investigation into the mental and emotional environment of a patient by a process varying from a sympathetic talk to a definite emotional catharsis is frequently loosely, though incorrectly, called psychoanalysis. The term really covers a detailed, highly complex and time-consuming method of examination, in which the greatest difficulty is the interpretation of the objective findings, and this interpretation is dependent to a great extent upon the contributions of the Freudian school to this vast subject. Regarding public interest in psychology, it is interesting to recall that Joseph Collins, the American neurologist and *litterateur*, when asked to give a talk on this topic to the girls' club of a large New York church, demurred, suggesting that other subjects might be more useful. The pastor, however, assured him that all the girls were reading psychology, and that he had good reason to think that the influence was one of harm rather than good, owing to their poor comprehension of such reading and poorer adaptation for the imbibition of such literary strong drink. This addiction to what Collins calls, in different metaphor, "mental fancy work", is just as rife today, and we cannot be sure that even the medical profession as a body can lay claim to any sound knowledge of psychology, let alone the highly technical procedure of psychoanalysis. Leo Kessell and Harold T. Hyman, of New York, have attempted to assay the psychoanalytical ore, to extract the therapeutic gold from it, and to find how valuable it may justly be considered in the actual handling of ailments that have a psychic basis, overt or covert.[1] At the outset it would appear that the gold extraction is accomplished in earnest, for psychoanalysis is so tedious and demands so great an output of time that the practitioner who specializes in this field, cannot handle many patients at one time. Consequently the number of patients who can afford to attend the analyst for three to six hours a week for well over a year, at a fee that is rarely less than three dollars an hour in New York, and often over ten dollars an hour, must be distinctly limited. The experience of these writers is not gained in the actual practice of psychoanalysis. They have referred apparently suitable patients to recognized and reputable psychoanalysts and they summarize the actual results of such treatment over a period of twelve years. Their summary is a frank and honest effort to reach the truth, and it cannot be said to be an enthusiastic advocacy of the method. Of thirty-three patients they find that bad results were obtained in half the cases, meaning that this moiety of the subjects were committed to institutions for definite psychoses, or otherwise showed an unfavourable outcome. For this, of course, it would not be fair to blame the method of treatment in all, if any, of the cases, but it must be remembered that the conscientious analyst will not accept patients for treatment with a frank and advanced psychosis. The patients treated comprised those with doubtful or early signs of psychosis, those with maladjustments and behaviour problems, those with neurosis, and those with functional visceral disorders. Among the remaining half of the series there were six complete failures. In four other instances the patients attained a good result, but this was qualified by an alteration in their state or behaviour, for example, in the direction of sexual liberation; the result here cannot be regarded as solely due to the analysis. The group of completely satisfactory cases comprised thirteen patients, about 40% of the total, and in some of these the authors are satisfied that the results were specific and could not have been attained by other means. All these patients were not completely relieved, however, and the number that could be recorded as unreservedly successful was probably not 20% of the total.

Thus it will be seen that the labours were great, the cost high, and the fruits of toil and expense relatively few. Moreover, results were seldom attained except in the young; over the age of forty little, if anything, can be expected. The patient, too, needs to be intelligent and acute of mind, a proviso that unhappily rules out a not inconsiderable proportion of those who form the doctor's public. Kessell and Hyman sum up the situation by saying: "Despite our receptive attitude towards psychoanalysis as a form of therapy in twelve years we have seen only a handful of patients who have benefited from their experiences."

[1] *The Journal of the American Medical Association*, November 18, 1933.

In an editorial comment on this article it is pointed out that while in 1930 the Berlin Psycho-Analytic Institute carefully valued the results obtained in 721 cases handled in ten years by its staff, this particular estimate was made by psycho-analysts. In the reviewed article, however, the evaluation was judicially made by sympathetic and impartial observers, and in this connexion reference is made to the report of the Commission of the British Medical Association of a few years ago.

Of course, in justice to psychoanalysis, it must be realized that its value is not to be solely measured by its therapeutic results. There still remains the research side of its activities, in which there is hope of useful work: for example, if light can be thrown on the mechanism of schizophrenia, advance will have been made. Also, Kessell and Hyman report only a handful of cases, and even in these their method of therapeutic evaluation might here and there be questioned. But it is an interesting communication and gives some idea of the prognosis in the groups of cases treated; also it serves to emphasize that it is only a very small field that can be covered by the psychoanalytical method.

ASCORBIC ACID AND SCURVY.

The water-soluble, antiscorbutic vitamin *C* is the most unstable of all the accessory food factors. It is readily destroyed by cooking, by boiling in the presence of oxygen, and even by drying. In the absence of oxygen it is more resistant to heat, but boiling with alkalis rapidly inactivates it. Even pasteurization of milk diminishes its activity. The body needs a constant supply of this vitamin, its storage capacity being almost negligible. An abundant supply at one time does not provide for the future. Birds, the rat and the prairie dog are immune to scurvy; but the livers of rats fed on a vitamin *C*-deficient diet will cure scurvy in guinea-pigs. Evidently the rat manufactures vitamin *C* itself irrespective of its diet. About seven years ago Bezssonov prepared a concentrated preparation of this vitamin and claimed to have obtained a substance, free from fats and proteins, which, in daily doses of 0·1 gramme, actively protected guinea-pigs from scurvy. Rygh considered that the alkaloid narcotine was converted into vitamin *C* by irradiation. Its absorption spectrum was investigated and examinations were made of the effects of light on a concentrate of vitamin *C*, narcotine and hexuronic acid. It has now been established that vitamin *C* is not an activated form of narcotine. Szent-Györgyi has identified vitamin *C* with ascorbic acid (one of the hexuronic acids). This ascorbic acid was isolated from the suprarenal cortex and subsequently by Svirbely and Szent-Györgyi in concentrated form from Hungarian red pepper (paprika). Its absorption spectrum was investigated. Crystals of hexuronic acid show a large flat cell, which, together with a strong negative birefringence, indicates ring molecules. It was claimed that ascorbic acid prevented scurvy in susceptible animals. It is not definitely established that ascorbic acid is actually vitamin *C*, but it may bear the same relationship to it as does carotene to vitamin *A*. It is not possible to say to what extent ascorbic acid (hexuronic acid) can be identified chemically and physically in the natural sources of vitamin *C*, such as green leaves, the juices of citrus fruits and the suprarenal cortex. The Tillman-Harris test for ascorbic acid is a quantitative method, based on the use of Tillman's indophenol indicator, and has been improved by preliminary separation of reducing substances which interfere with the test. The test is valuable for investigation, but failed in connexion with malt extract. By its means vitamin *C* was detected in the lens of the eye.

Possibly the earliest case reported in the literature of ascorbic acid being used curatively in scurvy was by P. Schultzer.[1] A man, aged sixty-eight years, with alcoholic gastritis and achylia, had been on a diet very deficient in vitamin *C*. He had definite evidence of scurvy, which subsided rapidly with intravenous injections of 40 milligrammes of ascorbic acid. Elisabeth Svensgaard reports two cases of infantile scurvy treated with ascorbic acid.[2] A male, aged fifteen months, suffered both from scurvy and rickets. The child had been at first fed on milk mixtures, and from the age of six months had been kept almost solely on rice gruel. The child was placed on oatmeal gruel alone as food, with "Cevita" (ascorbic acid) 30 milligrammes daily by mouth. This treatment was continued for two weeks. Within three days improvement was striking and the scurvy entirely disappeared. Tenderness to touch rapidly subsided, and the child, who previously refused to move spontaneously, crawled about the bed. The gums became normal and the child bright and happy. The other child was a female, aged ten months. Two months before admission to hospital the child was cross and peevish, wincing on being touched. She refused to stand on her feet, even when supported. The gums were tender and bluish. For the first month she was breast-fed. Then she was given milk mixtures, then only oatmeal gruel boiled in milk. She had no fruit, vegetables or cod liver oil. She was placed solely on oatmeal gruel, boiled in water. Ascorbic acid (30 milligrammes by mouth) was given daily. Within five days tenderness to touch had subsided and the gum lesions almost disappeared. In a week all symptoms of scurvy had vanished. Both of these children recovered in a few days, although they were kept on a diet lacking vitamin *C*. Ascorbic acid was the only treatment.

If these results are confirmed, the prophylaxis and treatment of scurvy will be enormously simplified. Long voyages in sailing vessels, polar and other explorations, and settlement in the remote parts of the earth will be deprived of much of their disabilities.

[1] *The Lancet*, September 9, 1933.
[2] *The Lancet*, January 6, 1934.

Abstracts from Current Medical Literature.

RADIOLOGY.

Trauma and Diseases of the Chest.

L. R. SANTE (*American Journal of Roentgenology*, July, 1933) states that traumatic pleurisy is due to an irritation of the pleura produced by trauma and has no connexion with bacterial infection. Pleural effusion rarely develops, and when it does, the fluid is a transudate. Unless infection is actually carried into the pleural cavity by the effect of trauma, there is little likelihood of bacterial infection occurring. That tuberculosis ever develops in a previously healthy person as a direct result of trauma seems very doubtful.

X Ray Examination of the Digestive Tracts of Infants and Children.

LEON T. LE WALD (*Radiology*, September, 1933) describes the typical Röntgen findings of pylorospasm. On the administration of an opaque meal the pyloric end of the stomach fails to fill. After several minutes this region fills out, but nothing passes through the pylorus. If skiagrams are now made at intervals of fifteen to twenty minutes, no food will be observed to pass through the pylorus until, perhaps, after from forty-five minutes to an hour. From this time on the pylorus may function in a fairly regular manner, so that at the end of the average emptying time only a slight retention may be present. In other cases the pyloric aperture becomes spasmodically closed and does not relax again for another interval of from forty-five minutes to an hour. In this type of case there will usually be a large gastric retention.

Calcification in Aortic and Mitral Valves.

MERRILL C. SOSMAN AND PAUL H. WOSIKA (*American Journal of Roentgenology*, September, 1933) state that it is possible to visualize calcified heart valves during life both by fluoroscopic and radiographic examination. The calcified valves are best demonstrated by fluoroscopy and appear as dark areas in the heart shadow which show a dancing movement with each heart beat. The patient should be rotated so that the right shoulder is 15° to 20° forward—a modified right antero-oblique position. When films are required, the exposure must be very rapid, at least one-twentieth of a second, and the voltage must be sufficient to penetrate the heart shadow. The calcified valves uniformly demonstrate a high degree of mobility, moving towards the apex with systole and returning with diastole. Most of the calcified aortic valves were located at the left border of the spine in the direct postero-anterior view, usually being hidden in whole or in part by the shadow of the spine and mediastinal contents. Antero-posteriorly they are usually deep in the heart, about midway or just behind the centre, as seen in an exaggerated left antero-oblique position. By comparison the calcified mitral valves are usually seen well to the left of the spine in the direct postero-anterior view and lie in the posterior third of the heart shadow in the exaggerated left antero-oblique view, sometimes so close to the posterior wall that they are thought at first to be in the pericardium. This is particularly true of those cases in which the calcification is thought to be in the *annulus fibrosus* of the mitral valve. In the markedly enlarged hearts of mitral disease the calcified valve may be well to the left of the spine and much more toward the apex of the heart shadow than one would expect, suggesting that by far the greater part of the heart shadow is formed by the enormously dilated auricles. Calcification in the mitral valve leaflets is accompanied by definite signs of mitral stenosis. Calcification in the *annulus fibrosus* of the mitral valve may be entirely unrelated to disease of the cusps and produce no cardiac symptoms.

Differential Diagnosis of Atelectasis.

W. F. MANGES AND J. T. FARRELL (*American Journal of Roentgenology*, October, 1933) consider that the radiographic signs of atelectasis are important in the differentiation of pulmonary abscess secondary to neoplasm from abscess unassociated with neoplasm. In pulmonary abscess uncomplicated by newgrowth, displacement phenomena are absent unless there is pleural involvement; then the diaphragm alone is affected, the mediastinal structures remaining in their normal position. In abscess following bronchial newgrowth there is practically always displacement of the thoracic organs towards the side of involvement. The difference in displacement is probably accounted for by the difference in the mode of development of the two varieties of abscess. Acute non-neoplastic abscess follows entrance of infected material into the alveoli in an amount great enough to set up a suppurative focus, but not large enough to produce bronchial obstruction and subsequent atelectasis. Destruction extends centrifugally from a central point of origin. Extension of the tumour from its primary site in the bronchus into the atelectatic pulmonary tissue converts the pulmonary tissue into a neoplastic mass. Necrosis of the centre of the tumour and bacterial invasion bring about abscess and cavity formation. The collapsed lung remains diminished in size because the neoplastic tissue cannot expand, and as a consequence the thoracic viscera are held in their position of displacement. In some patients with asthma atelectasis of sudden occurrence is responsible for attacks of dyspnœa. The condition is probably of more frequent occurrence than is generally realized. Plugging of a bronchus with mucus may well be the causative factor in these allergic patients. The radiographic findings are those of classical atelectasis, complete opacity of the collapsed lobes and displacement of the heart and mediastinal structures toward this side of the chest. With reexpansion dyspnœa lessens, the lungs assume their usual emphysematous appearance, and the mediastinal structures return to their normal positions.

Malignant Disease and Chronic Prepyloric Gastric Ulceration.

A. O. HAMPTON (*American Journal of Roentgenology*, October, 1933) emphasizes the importance of the anatomical location of gastric ulcerations when considering the possibility of malignant disease. Observations are reported which seem to indicate that chronic indurated prepyloric ulcers should be considered and treated as malignant ulcers. The prepyloric area is sharply limited to that portion of the stomach just proximal to the pyloric valve which is 2·5 centimetres (one inch) in length. No part of the pyloric valve is included in the prepyloric area. Apart from actual measurement, this portion of the stomach may be identified during radioscopy by its canalization at the height of peristaltic activity. The relative proportion of benign prepyloric ulcers to carcinomata in the same area of the stomach is as 1 to 12. That is, in a given case, when definite pathological change is localized to the prepyloric area, the chances of the lesion being carcinoma are twelve times as great as are the probabilities of its being a benign ulcer. Eight patients were followed up in whom posterior gastro-enterostomy had been done without any other operative procedure for the treatment of what was thought to be benign chronic prepyloric ulcerations. All of these patients returned to hospital within an average period of two years, presenting unmistakable evidence of advanced carcinoma of the stomach.

X Ray Examination in Polypoid Tumours of the Larynx.

W. H. MCGEHEE (*American Journal of Roentgenoology*, October, 1933) calls attention to the fact that polypoid tumours of the larynx can be demonstrated on the skiagrams when they are so located as to be seen in profile. Most of these tumours arise from the anterior wall of the larynx, either just above or just below the anterior commissure, and they are usually benign. They are characterized by localized bulgings encroaching on the lumen of the larynx. X ray examination of the neck is very valuable in children and adults who will not cooperate in the examination.

Lipiodol in Bronchography.

J. BURNS ANDERSON AND H. MCLEOD RIGGINS (*American Journal of Roentgenology*, December, 1933) state that heating lipiodol above body temperature lowers its viscosity and makes

the injection easier for the operator; but there are objections to this. A very freely flowing oil enters the pulmonary alveoli more readily, thus tending to obscure the radiological distinctness of the bronchi. Furthermore, oil in the alveoli is not easily expelled and may remain trapped there indefinitely, which is a disadvantage. It is better, therefore, not to heat lipiodol, as is often done, but to use it at a temperature of 18·3° to 23·8° C. (65° to 75° F.). After the skiagrams are made, the patient hangs over a table, without coughing, to drain his bronchi. Most of the oil will flow out in ten or fifteen minutes. The importance of this postural drainage cannot be stressed too much.

PHYSICAL THERAPY.

Deep X Ray Therapy in Arthritis Deformans.

G. VON PANNEWITZ (*Deutsche Medizinische Wochenschrift*, April 21, 1933) reviews a series of 1,507 patients with *arthritis deformans* treated over a period of six and a half years with deep X ray therapy. He found that the age of the patient had no effect on the final result of treatment. The site of the disease was important: improvement occurred in 76% of spine cases, in 87% of joints of the lower extremity, and in 89% of the upper extremity joints. The longer the duration of the disease, the worse the prognosis, whilst the severity of the disease was of less importance. Thus in patients who had symptoms for one month, 85% were benefited, whilst of those who had symptoms for one year, 50% only were benefited. Early diagnosis and treatment are therefore essential. In 140 cases under treatment and constant observation it was found radiologically that in 65% of the cases the joint changes remained stationary, whereas in the other cases the steophytes *et cetera* increased in size. Except in one instance, no change in the nature of an improved X ray picture occurred. Clinically, symptoms do not run parallel with the X ray findings. The effective dose was variable and adapted for each part and joint.

Treatment of Osteoarthritis by X Rays.

S. GILBERT SCOTT AND F. HERNAMAN-JOHNSON (*Medical Press and Circular*, October 4, 1933) state that X rays are most valuable in the treatment of certain types of arthritis, but success necessitates correct diagnosis. First of all the "rheumatoid" group must be excluded. Cases of the rheumatoid type, in the authors' experience, are not benefited by X ray treatment. In contrast to the above there is the monarticular type, which is found oftener in men and which as a rule does not give serious trouble until late middle age. Hips, spine and knees are the usual sites. The history is one of slow onset of symptoms, except in those cases in which there has been an exciting trauma. Radiographically there are numerous osteophytes, and in the case of the hip the femur may be partially extruded from the acetabulum. Cases which conform to the above description usually do well under X ray treatment. When there are few osteophytes and when the interarticular cartilage is still more or less intact, a complete symptomatic cure may be expected. Considerable deformity is not, however, necessarily any bar to a striking improvement, and this may come about without even change in the radiographic appearances. The X ray treatment apparently enables the patient to make full use of whatever movement is mechanically permitted; this is always much greater than the patient originally had, as the range of movement is in many cases limited to a great extent by pain and muscular spasm. Several cases are quoted, but no details of the technique of treatment are given. The authors recall three of the well established therapeutic properties of X radiation: (i) It is analgesic; (ii) it produces congestion and inflammation; (iii) it has the power of causing the absorption of pathological fibrous tissue, as is demonstrated when it is applied to keloidal scars.

Effect of Radiation on Blood.

G. W. PHILLIPS (*The Lancet*, September 30, 1933) took six nurses who did not come into contact with radium or X rays and used them as controls. Blood counts were done from these people, and also from nine patients suffering from malignant disease who were under treatment with large doses of irradiation. The conclusions arrived at are that in no case following massive doses of radiation was an anæmia produced. The white cells were not materially altered in any way, and a leucopenia did not occur. A low lymphocyte count was found in most of the cases of malignant disease, but the effect of the large doses used was definitely not the production of a lymphopenia. The results of the investigation suggest, therefore, that no characteristic changes are produced in the blood pictures by large doses of radiation; also that the blood count *per se* should not limit the amount of irradiation that it is considered necessary to give.

Actinotherapy in Urogenital Tuberculosis.

A. LEQUEU, J. SAIDMAN AND S. AVRAM (*The British Journal of Physical Medicine*, May, 1933) use ultra-violet rays from the usual type of lamp and claim successful results in the treatment of tuberculous orchi-epididymitis, particularly in 105 patients not operated on. They suggest that actinotherapy is indicated in the first place, and an operation should be performed when ultra-violet rays fail. They show that the pain is acute in the early stages, but rapidly subsides after the first two treatments. The mass diminishes in size, the clear demarcation becomes visible between it and the testicle, which frees itself from all epididymal adhesions. Twenty to sixty treatments are required to reach this stage. Among seventy cases there were fifty-two cures and eighteen improvements. The sinuses occurring spontaneously take longer to heal. The authors also use this method of treatment for sinuses following stitch abscesses, and claim an additional improvement in bilateral renal tuberculosis.

The Use of Physical Therapy in Medicine.

LOUIS J. GELBER (*Radiology*, September, 1933), in an article on some applications of physical therapy in medicine, deals with arthritis, whether traumatic, atrophic, hypertrophic, or infectious in character. He states that he collected all gonorrhœal arthritic patients from the wards after other methods of treatment had failed and sent 85% of them home 100% efficient after treating them with deep therapy, infra-red rays, diathermy or static massage, as indicated by the condition. Electro-surgery has come to stay, and electro-desiccation and electro-coagulation both have their uses. The author recommends diathermy or galvanism in localized muscular inability or dysfunction of the colon. He suggests early cooperation between the surgeon and physiotherapist in post-fracture work. In treating gynæcological disease by diathermy the abdomino-vaginal method is given precedence. An eight by ten electrode is placed over the abdomen and another in the vagina behind the cervix; from 1,200 to 1,600 milliampères are used. The contraindications given are acute infections, ovarian cysts or tumours, and menstrual periods.

Sycosis and X Rays.

A. MARIN (*Canadian Medical Association Journal*, June, 1933) states that sycosis is very obstinate, sometimes developing over a number of years and leading to permanent alopecia. The fact that the infection is so deeply seated explains its obstinacy. External applications do not reach it and the same may be said of ultra-violet ray treatments. Extraction of hairs is a lengthy and painful procedure, whilst with ionization the proportion of cures is not high. Recourse therefore must be had to radiotherapy. The author varies his technique and dosage according to severity and duration of the disease. In mild cases only about one-quarter of an erythema dose is given, whilst in refractory cases he gives as much as half an erythema dose every three weeks for four or five months. In conclusion, the author states that the treatment of sycosis by X rays is in most cases the only method of treatment that is effective and that it cures patients more rapidly and at a cost less than that of other forms of treatment.

Special Report.

SYMPATHETIC RAMISECTION.

THE following is the report of the Victorian Committee appointed by the Royal Australasian College of Surgeons to investigate the surgery of the sympathetic nervous system.

As the result of the adverse conclusions arrived at by a number of English orthopædic surgeons upon the value of the operation of sympathetic ramisectomy in cases of spastic paralysis, and especially in view of the claims of Dr. Norman Royle that this operation was of value in such conditions, the Editor of THE MEDICAL JOURNAL OF AUSTRALIA requested the Royal Australasian College of Surgeons to undertake an investigation of the subject in all its aspects. A subcommittee of the College was therefore appointed in Melbourne to examine the evidence available. Power was given this committee to coopt other members of the medical profession, and scientists who were considered best qualified to assist in the investigation. The following were appointed as members of the subcommittee for Victoria: A. E. Coates, J. B. Colquhoun, R. M. Downes, C. W. B. Littlejohn, Fay Maclure, Dr. H. F. Maudsley, Sir Richard Stawell, H. Douglas Stephens, Dr. O. W. Tiegs (coopted), H. C. Trumble, Professor F. Wood-Jones.

Dr. A. E. Coates acted as Secretary to the Committee and Professor Wood-Jones as Chairman.

Meetings of the subcommittee were held in 1931, 1932 and 1933. In all, seven meetings were held. The leading surgeons of Melbourne and country centres in Victoria were circularized and asked to furnish the names of patients who had been submitted to sympathectomy for spastic conditions, and permission was obtained to examine these patients. Dr. Royle kindly forwarded the names of patients residing in Victoria upon whom he had operated for spastic types of paralysis. The records of the metropolitan hospitals were made available to the subcommittee, and one meeting was held at the Children's Hospital (Melbourne) and one at the Alfred Hospital (Melbourne). A few patients on whom ramisectomy was done for diseases other than spastic paralysis were also examined, but attention was devoted chiefly to conditions of spastic disease, since the data, both experimental and clinical, was more complete in regard to this condition.

During the period 1931-1933 there appeared in medical periodicals publications dealing with the subject of sympathetic ramisection and the treatment of spastic paralysis, and special reference was made in the meetings of the subcommittee to: (1) the article by A. E. Coates and O. W. Tiegs (Melbourne) in *The Australian and New Zealand Journal of Surgery* (March, 1931) with subsequent personal communications in reference to special experimental work; (2) an article by Gilbert Phillips, of Sydney, in *Brain*, Volume LIV, 1931; (3) an article by N. D. Royle (Sydney) in *The British Medical Journal* (June 11, 1932), in which he states that he has modified his views concerning the basis of any change which occurs after ramisectomy. Dr. Royle's present contention that unilateral ramisectomy involving the sympathetic neural supply to the head produces special changes in the circulation of the brain requires very critical consideration based upon special experimental investigation. It was considered that this was outside the scope of the inquiry by the present subcommittee.

The chief purpose of the work of the subcommittee was the examination of the clinical results of ramisection as originally carried out by Dr. Royle and suggested by the late Professor Hunter. These clinical results have been disappointing as regards the relief to any degree of the types of spasticity in the cases examined by members of the subcommittee or of the cases reported to the subcommittee. The subcommittee deliberated upon the following aspects of the subject under review:

1. The anatomical evidence for the existence of a sympathetic supply to striated muscle. The literature, now very extensive, was summarized and reported on by various members, and it has been commented on in the report by Dr. O. W. Tiegs.

2. Physiological evidence. The accumulated literature and the records of experimental work were examined and recorded. Dr. Tiegs' paper covers the most important aspects of this field.

3. Experimental evidence was discussed; the original work of Hunter and Royle and the subsequent work by a number of observers were reviewed.

4. The clinical evidence. In reviewing the clinical features that followed the operation of ramisectomy, the most constant result was a condition of vasodilatation leading to increased warmth in the affected limb. Thirty-nine (39) cases in all were considered and, where possible, personally examined. (*a*) Of cases operated on by Dr. Royle, three were available for examination in detail, four others were reported on by the attending medical man or the patient's relatives. [See reports attached (Appendix *A* 1).] (*b*) Cases operated on by surgeons in Melbourne were examined and reviewed. A careful history of the conditions present before operation was obtained. The condition of the patients at the time of examination was noted and an attempt made to estimate the improvement, if any, resulting from the operation.

In the three cases operated on by Dr. Royle, examination by the subcommittee and interrogation elicited no evidence of any improvement attributable to the operation of ramisectomy. Four other cases mentioned by Dr. Royle were communicated with by letter, but were not personally examined. With one exception, the replies received were unfavourable as regards any improvement. (Appendix *A* 2, Dr. Royle's cases.)

Dr. H. Douglas Stephens reported eight (8) cases on whom he had performed ramisectomy for spastic disease. No improvement was noted by Dr. Stephens following the operation, nor could the subcommittee find any sign of lessened spasticity in several of these cases examined at the Children's Hospital (Melbourne). (See Appendix *B*.)

Dr. Rupert Downes reported four cases of ramisectomy for spastic disease. In one case the mother thought there had been some improvement in the seven years that have elapsed since operation. The subcommittee examined this boy and agreed that slight improvement had occurred since the operation. (See Appendix *C*.)

Dr. Hugh Trumble showed three cases of spastic disease and one case of post-encephalitic Parkinsonism, subjected to ramisectomy. No improvement occurred in any case, although chilblains cleared up in one case, apparently due to vascular changes in the limb. (See Appendix *D*.)

Dr. C. W. B. Littlejohn reported that he had operated on twelve cases of spastic disease by ramisectomy. Seven of the cases were operated on in England and examined by several prominent physicians and physiologists there. They could find no evidence of any change in the condition of the limbs, apart from vasodilatation. In none of Dr. Littlejohn's cases had there been any lasting benefit. (See Appendix *E*.)

Sir R. R. Stawell reported fully on two cases in which he had recommended the operation of ramisectomy for spastic disease. No improvement had occurred in either of them. (See Appendix *F*.)

Dr. W. Allan Hailes reported on two cases of spastic disease upon whom he had performed the operation of ramisectomy. No improvement had resulted in either case. (See Appendix *G*.)

In collaboration with Dr. O. W. Tiegs, the experimental work on the sympathetic nervous system has been reviewed by the subcommittee. The summary of the literature to date indicates:

1. There is no definite anatomical evidence of a dual innervation of striated muscle. There is much evidence to the contrary.

2. There is no physiological evidence of a control of muscle tone by the sympathetic nervous system.

3. There is evidence suggesting a relation of the sympathetic nervous system to the fatiguability of muscle. Recent work seems to show that there may be a humoral origin for the phenomenon. The absence of any effect of sympathectomy in tetanized muscle seems to exclude this procedure as being of any importance in the possible relief of spasm.

The results of the operation were reviewed in thirty-nine cases. Fourteen cases were carefully examined personally by the entire subcommittee at their meetings. No case showed any evidence of improvement in the spastic condition following ramisectomy.

The remaining twenty-five (25) cases were reported on by members or by practitioners as mentioned. In two of these reported cases a subjective improvement was noted and in one a slight objective improvement occurred in a boy of fourteen years seven years after operation.

The subcommittee is of opinion that any improvement which may occur in cases of spastic disease subjected to the operation of ramisectomy alone is: (*a*) psychic, together with (*b*) an appreciation of a warmer limb due to vascular changes, and that there is some danger of ascribing to the operation of ramisectomy certain improvements in the patient's condition due in reality to ancillary measures, such as tenotomies, massage, rest in bed *et cetera*, incidental upon the main procedure.

Report on the Anatomical and Physical Evidence for the Sympathetic Innervation of Skeletal Muscles by Dr. O. W. Tiegs.

The following statements are based on investigations which I have carried out during the last four years in collaboration with Dr. A. E. Coates at Melbourne University, with Dr. A. B. Corkill, of the Baker Institute, and partly alone. They were directed towards determining whether sympathetic nerves had any influence on skeletal muscle—direct or indirect—and, if so, how that influence was exerted. The investigations are as yet incomplete; but as far as they go they have, I think, some bearing on the problem of the relation of the sympathetic nervous system to spasticity in man. Hunter and Royle realized that the operation of ramisection must find its justification in clear-cut animal experiments, and for this purpose performed a series of, for the most part, well planned tests to verify their hypothesis. Most of these experiments were repeated by Dr. Coates and myself, with as much care as possible, but we were forced to conclude that Hunter and Royle had been mistaken. These experiments may be summarized as follows:

(1) *Effect of Sympathectomy on the Posture of the Limb of the Goat.*

(*a*) In the original experiments of Royle it was stated (THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1924, page 77) that a sympathectomized hind limb was unable to maintain the attitude of extension that is usually assumed when the animal is laid on its back; the operated leg was stated to become flaccid, whilst the control leg maintained a rigid extension. In our own experiments we were quite unable to confirm this; frequently both legs would be flexed, or both be extended, and they would maintain this attitude for many minutes. At times one leg would flex, the other extend, but it was just as often the unoperated as the operated leg that was flexed. In one animal only did we consistently observe the effect described by Royle and, in this case, it was almost certainly ascribable to trauma, since a towel had to be left in the wound to stop a severe hæmorrhage that had occurred. (Reference, Coates and Tiegs, *Australian Journal of Experimental Biology and Medical Science*, Volume V, 1928, page 11; *ibidem*, Volume VII, 1930, page 37.)

(*b*) *The Effect of Decerebrate Rigidity.* Royle reported an inability of the operated leg to maintain the attitude of extension that ensues on decerebration. We did not perform any decerebration tests. They have, however, been carried out by Mortensen, Friedbacher and Quade (*Proceedings of the Society of Experimental Biology and Medicine*, Volume XXV, 1928, page 757) on the goat, and by numerous others on cats and dogs, and invariably with negative results.

(*c*) *Effect on the Knee Jerk.* It was claimed by Hunter (Lectures, *The British Medical Journal*, 1925, page 197) that sympathectomy had a profound effect on the knee jerk. It was stated that the knee jerk of the operated limb was short and sharp, that of the normal limb much more prolonged. In our experiments, on the contrary, the operation had no perceptible effect on the knee jerk.

(*d*) *Action of Ephedrine.* Royle (THE MEDICAL JOURNAL OF AUSTRALIA, June, 1929, page 740) sought to strengthen his argument by experiments with ephedrine. He reported that this "sympathetic stimulant", when injected into a goat, produced profound sympathetic hypertonus (that is, rigid extension) in the normal leg, but failed to produce that effect if the sympathetic had been previously cut and allowed to degenerate. The planning of the experiment is, of course, physiologically unsound, for "sympathicomimetic" drugs, as is well known, act not on the sympathetic nerve ending, but on the tissue innervated, even after sympathetic nerves have degenerated. Dr. Coates and I repeated this experiment with negative results (Coates and Tiegs, *Australian Journal of Experimental Biology and Medical Science*, Volume VII, 1930, page 37).

(2) *Experiments on Birds.*

Hunter's experiments on the posture of the bird's wing were particularly ingenious. It was stated: (i) That division of the preganglionic sympathetic fibres to the wing caused a droop in the wing. (ii) Desensitization of the wing, produced by dorsal root section, abolished the somatic reflex arc, so that the wing, whilst retaining its sympathetic postural tone, was incapable of voluntary movement. (iii) And that subsequent division of the preganglionic fibres to a wing thus desensitized left it completely paralysed. As the experiments would have afforded such a brilliant demonstration of the Hunter-Royle theory, we repeated them, taking the utmost precautions in our operative technique (Coates and Tiegs, *Australian Journal of Experimental Biology and Medical Science*, Volume V, 1928, page 11). We found: (i) that division of preganglionic fibres produced a droop in the wing only when adjacent spinal nerves were injured; (ii) that voluntary movements in the desensitized wing were not abolished; (iii) that the alleged sympathetic postural tone of the desensitized wing was not abolished by subsequent division of the preganglionic fibres. In fact, the postural tone that undoubtedly does persist, as Hunter found, when the dorsal roots are cut, is due to impulses emerging through the ventral roots of the brachial plexus, which he had not cut, failing, as he did, to realize that the bird's wing, unlike the mammalian limb, cannot be deprived of tonus simply by cutting its dorsal nerve roots.

In later experiments I found (Tiegs, *American Journal of Physiology*, Volume XCVIII, 1931, page 547) that the bird's wing deprived of its sympathetic supply did not show any trace of a droop, even when the possibility of compensatory action of somatic motor nerves had been abolished by dorsal root section.

The general conclusion drawn from these experiments is that the limb musculature retains its postural action in the absence of sympathetic nerves; even under circumstances (as in the bird's wing) where the possibility of compensation by spinal nerves is excluded.

Dr. Royle criticized our results (THE MEDICAL JOURNAL OF AUSTRALIA, February, 1928, page 257) on the ground that we could not have completely removed the whole abdominal sympathetic trunk in our operations on goats and that undetected fragments of sympathetic ganglia, left behind, obscured the effect of the operation. To this we would reply that we undoubtedly removed the greater part of the abdominal sympathetic trunk in our animals, and that even if undetected fragments were left behind, as Dr. Royle asserts, they could surely not take over the whole of the function of the rest of the abdominal sympathetic trunk and thereby obscure the effects of its removal. In our turn, we would point out that a complete or even partial destruction of the sympathetic chain not followed

by a droop in the limb is necessarily a better experiment than one in which a droop occurs; for such a droop can be produced by accidental injury to adjacent spinal nerves, an injury that is not easily avoided.

(3) *Sympathetic Innervation of Skeletal Muscle.*

At the time we commenced our experiments we believed in the existence of a double innervation of skeletal muscle. This doctrine is mainly the outcome of the researches of Boeke and Agduhr. Most of Boeke's work has been performed on the extrinsic eye muscles; Agduhr's on the cat's interossei. Concerning Boeke's experiments, it now appears that he chose particularly unfavourable material and did not allow sufficiently for complete degeneration of all somatic nerves. His experiments have been repeated with negative results by Hines (*American Journal of Anatomy*, Volume XLVII, 1931, page 1), by Wollard (*Journal of Anatomy*, Volume LXV, 1931, page 224), and by Wilkinson (*Journal of Comparative Neurology*, Volume LI, 1930, page 129). We have not repeated these experiments ourselves, for if the histological conclusions are to be applicable to limb muscle, then these nerves should be histologically demonstrable in them also. Agduhr had indeed claimed that this was the case. We repeated this histological work on the foot muscles of the dog. When all somatic nerves were degenerated and the tissue subsequently prepared by the Bielschowsky technique, the only intact nerves were certain coarse sensory nerves and the sympathetic nerves to the blood vessels (Coates and Tiegs, *Australian Journal of Experimental Biology and Medical Science*, Volume VIII, 1931, page 99). No sympathetic nerves were found on the muscle tissue. In explanation of Agduhr's apparently positive results it may be pointed out that Wilkinson (THE MEDICAL JOURNAL OF AUSTRALIA, 1929, page 768), who examined Agduhr's own preparation, found scattered coarse medullated nerve fibres in them, with large end-plates on the muscle fibres, indicating, of course, that Agduhr's surgical procedure had been defective.

In this connexion the muscle of reptiles is of great interest, for here we can regularly display two kinds of nerve fibres ending on the striated muscle fibres—coarse medullated and fine non-medullated or only weakly medullated fibres. The latter have been known ever since Tschiriew described them in 1879 and have been variously described as immature nerves, sensory nerves and, recently, as sympathetic nerves (Kulchitsky). It was Kulchitsky's work on python muscle that first attracted Hunter's interest, and Kulchitsky's preparations, seen by Hunter while he was in London, provided the histological basis of his theory.

To determine the origin of these nerves I performed degeneration tests on a python and on a large local lizard (Egernia). It soon became apparent in these experiments that the nerves were not sympathetic at all, as Hunter had believed, but were in reality somatic motor nerves (Tiegs, *Journal of Anatomy*, Volume LXVI, 1932, page 300). Some of the preparations on which these findings were based were exhibited at a meeting of the Biological Sciences Club at the University about a year ago, when several members of your Committee were present.

It would appear, therefore, that at present we have no indisputable evidence that sympathetic nerves directly innervate the skeletal muscle fibres. Apparently they supply only its blood vessels. It may be argued that we have failed to stain the nerves; but it would seem impossible that a plexus of sympathetic nerves, not related to blood vessels, should exist and escape our staining methods when those same methods stain the nerves on blood vessels so well.

(4) *Relation of the Sympathetic Nerves to Fatigue of Muscle.*

In 1923 Orbeli, a pupil of Pawlow, reported experiments from which it appeared that sympathetic nerves had the power of relieving the fatigue of skeletal muscle. Dr. Coates and I (Coates and Tiegs, *Australian Journal of Experimental Biology and Medical Science*, Volume V, 1928, page 11) also observed quite unexpectedly that goat muscle deprived of its sympathetic supply for many months fatigued much more easily than did normal muscle; but our experiments were indecisive, since prolonged absence of the sympathetic may have had an injurious effect on the muscle. More recently, Maibach (*Zeitschrift für Biologie*, Volume LXXXVIII, 1928, page 207) has confirmed Orbeli's experiments on the frog; and Miss A. Baetjer has confirmed them on the cat (*American Journal of Physiology*, Volume XCIII, 1930, page 41).

For the past nine months Dr. Corkill and I have been investigating this question. Although our work is not yet complete, we have determined the following facts:

(i) Stimulation of the sympathetic nerves to isolated frog muscle has the effect of markedly relieving fatigue in that muscle.

(ii) The effect is not due to changes in the local blood supply.

(iii) It is only when the fatigue is being produced by indirect stimulation of muscle that the Orbeli effect appears; muscle stimulated directly does not show it. The site of action of the sympathetic effect therefore appears to be the myo-neural junction.

(iv) All the effects of sympathetic nerve stimulation may be imitated, even in detail, by perfusion of adrenaline into the muscle. We have not yet undertaken corresponding experiments on mammals.

These conclusions seem to contradict the statement above made that sympathetic nerves are confined to the blood vessels of the muscle. But some recent work on the peripheral action of the sympathetic offers a possible escape from this dilemma.

(i) Loewy has produced definite evidence to show that the peripheral action of the cardio-accelerator nerve is humoral. (ii) Dale and Gaddum have recorded the observation that stimulation of the sympathetic nerves to blood vessels of muscle produces certain local effects that can best be explained on the assumption that a chemical substance (with adrenaline-like properties) is liberated in the muscle. (iii) W. B. Cannon finds that stimulation of sympathetic nerves to the cat's tail causes the formation of a chemical substance which, passing into the blood, can accelerate the action of the denervated heart. The general implication of all this work is that a peripheral sympathetic hormone (sympathin of Cannon) exists and that it is in fact adrenaline.

If, then, such a hormone is liberated in sufficient concentration in the muscle tissue by the vascular nerves, it might well produce the striking relief of fatigue that we observe experimentally; whether this be the explanation of the Orbeli phenomenon is not yet known, but Dr. Corkill and I are at present investigating this possibility. This unexpected action of the sympathetic nerves raises another problem to which no definite answer can yet be given: Since the predominant effect of sympathetic stimulation is vaso-constriction, would we expect the sympathetic nerves to come into play at the time when the muscle is presumably most in need of a good blood supply, namely, during fatigue? Vaso-constriction would accelerate fatigue. Some recent experiments by H. Wastl (*Journal of Physiology*, Volume LX, 1925, page 109; *Pflüger's Archiv*, Volume CCXIX, 1928, page 337) show that sympathetic stimulation or adrenaline injection to an active muscle (cat) may in fact depress its activity markedly. On the other hand, experiments by A. Baetjer (*American Journal of Physiology*, Volume XCIII, 1930, page 31) show that sympathetic stimulation, under what are apparently similar conditions, revives the muscle.

It seems to me that the powerful effect of sympathetic stimulation and of adrenaline on fatigued muscle that we have ourselves obtained must be of physiological significance; and yet sympathetic stimulation or adrenaline injection decreases the local blood supply and thereby tends to undo their own beneficial effects on muscle. The whole matter is at present very perplexing. Light may possibly be shed on it by a recent statement by Reid and Schneider (*Zeitschrift für Biologie*, Volume XCI, 1930, page 13) that adrenaline in physiological doses acts as a vaso-dilator when muscle is actively functioning, and not as a vaso-constrictor. It may be that sympathetic stimulation at

times acts in a like manner. But the whole matter requires investigation before a definite answer can be given.

Whilst there can no longer be any doubt that the sympathetic system does have an influence on skeletal muscle, it remains uncertain whether such an effect comes into play in the maintenance of postural tone. It might be argued that sympathectomy, by rendering a muscle more easily fatiguable, might thereby have a beneficial effect by counteracting excessive hypertonus. I do not think that this argument can carry much weight, for conditions of experimentally produced hypertonus are not perceptibly altered by removal of the sympathetic. For instance, Dr. Coates and I found that the profound hypertonus produced by injection of tetanus toxin was unaltered by sympathectomy. It seems to me, therefore, that the only demonstrable effect of sympathetic action (the relief of fatigue) cannot form a sound basis for the operation of ramisection for spastic condition of muscle, and anyone who would advocate its use on such grounds would have to explain why experimentally produced hypertonus in animals is entirely unaffected by it.

Conclusion.

The results of the committee's deliberations have indicated that the operation of sympathetic ramisection is of no permanent value in the treatment of spastic paralysis.

While due weight must be given to the evidence available and allowance made for erroneous conclusions arrived at by earlier workers in this field, the work of the late Professor Hunter and Dr. Norman Royle has not been for nought.

As in all human endeavour, premature enthusiasm is frequently curbed by the results which fail to meet early expectations. The pioneer work in a new field of surgery and the application of the physiological principles, many of which were known and forgotten, to the alleviation of human ills must surely be credited to the two Sydney investigators. Their work has stimulated a great deal of research which is still being prosecuted and which, doubtless, will bear fruit in the future. Keen students throughout the world have been inspired to earnest and careful work in the laboratory and in their clinical practice by the painstaking efforts of Hunter and Royle. Especial credit is due to Dr. Royle for the elaboration of the operative technique for the surgical interference with the sympathetic nerves. These operations cleverly devised and skilfully undertaken will perpetuate his memory with future generations of surgeons.

Appendix A 1.

Reports of Cases Operated on by Dr. Royle and Examined by the Committee.

(*a*) Mr. C., aged forty-nine years (Dr. McMahon's case). Twenty-seven years ago the fork of his bicycle broke. He struck his head on the left side. Ten days after injury he was operated on by Sir George Syme (Melbourne Hospital). Recovered consciousness, but suffered loss of power in right arm and leg. No loss of sensation. Temporary aphasia and dysarthria. Dragged right leg and flexed right arm. Could not feed himself. Operated on by Dr. N. Royle, 1926, Sydney. Two operations: first, lumbar, and later, cervical, ramisectomy. He thinks he swings the right leg less. No massage or exercise treatment. Right arm in position of pronation—flexion. No power of supination. Gait, swings right leg. Cannot turn easily; pivots on good leg.

Knee jerks.—Right, excessive, exaggerated; left, normal.

Plantars.—Right, extensor; left, flexor.

Handling feet.—Right, spastic, ankle clonus; left, flaccid.

Joint sense.—Left, right, normal.

Upper limbs.—Tendon jerks: Right, exaggerated; left, normal.

Sphincters.—Never had loss of power. (Had no erection after accident, but 16 months after operation erection occurred).

Contraction of thumb and fingers on right hand.—As in cases of U.M.N. lesions.

Incision.—Curved, renal type.

Temperature changes.—Right hand perspires, right leg does not. Right toes warmer than left. Right hand colder than left. Left leg half inch shorter than right.

(*b*) Mr. L., aged forty-six years (examined by Committee on December 18, 1931). Motor accident at Werribee, March 26, 1919. Brought to Melbourne Hospital. (Stammered before operation.) Operated on by Dr. Tymms at the Melbourne Hospital. Three weeks later under Sir George Syme in Mount Saint Evins Hospital, later under Dr. Sewell. He had massage. He could walk with assistance only. Dr. Sewell took him to Dr. Royle in 1924. He could walk moderately well with a stick at that time. The arm was weak. He had massage four years. Reeducative exercise. Three operations by Dr. Royle: (i) right cervical, (ii) right lumbar ramisectomy, and then (iii) tendon operation on hand.

As the result of operations by Dr. Royle, he thinks he is a little better in speech. He noticed no change in arm or leg following the operation, but he fancies the leg was slightly better. He thinks he is gradually improving now, but did not notice any marked improvement in the year following the operation.

Temperature.—The affected side feels colder than normal in winter, but hotter in summer.

Sphincters.—No change. Right hand perspires freely.

Gait.—Drags bad leg. Holds right arm in flexion.

On examination.—Knee jerks: right, exaggerated; left, normal. Both upper and lower limbs of the right side are rigid. Board-like rigidity. Right knee and ankle clonus. Plantars: Right, extensor; left, flexor.

(*c*) Case of M.H., girl, aged ten years. Operated on by Dr. Royle three years ago. Ramisectomy and tenotomy performed for spastic diplegia. Mentally defective. Examination by the subcommittee revealed a spastic deformity of both legs and feet. Adductor deformity of the thighs. A right subtemporal decompression had been performed when three years of age for the relief of fits. Dr. H.D.S. and Dr. G.A.P., who attended the child stated that no improvement had occurred as a result of ramisectomy. The child's grandmother stated that she thinks the child is better since the operation, but that the correction of the deformity by tenotomy was the most important cause of improvement.

The subcommittee could find no evidence of improvement as a result of ramisectomy in this case.

Appendix A 2.

Reports of Cases Operated on by Dr. Royle, but not Examined Personally by the Subcommittee.

R.C., aged three, a son of a medical man. The child suffered from spastic paralysis from birth. Right and left cervical ramisectomy and right lumbar ramisectomy performed by Dr. Royle. The child's father reports no improvement in the spastic condition.

I.R. (reported on by Dr. R.D.), male, aged eight years. Operated on at age of four for Little's disease by Dr. Royle. Examination by Dr. R.D., revealed a spastic condition of both legs. The mother reported that no benefit had been derived from the operation and was averse to any further operative interference.

Mrs. S.: A case of disseminated sclerosis with gross spasticity of lower limbs, treated by ramisectomy. L.M., her father, reported that no benefit had been derived from the operation.

Case in which mother of deceased patient was interviewed by Committee. Mrs. M., reporting on Miss M.M., thirteen years, deceased. Paralysis from birth. Both arms and leg. Could not use right arm or leg. Speech was affected. Mentally normal. Could not express herself. Three operations by Dr. Royle in Sydney. Bilateral ramisectomy and right cervical. Mrs. M. states that the patient could use her arms and legs better after the operation. She thinks she was almost able to walk at the time she died. Unsteady and restless before operation, and after operation she was quiet and composed. She

developed. She had half-hour morning and night exercises after operation. Before operation she crossed her legs. She could not sit up before operation, but after Dr. Royle's operation she could sit up. Her speech improved.

Appendix B.

Cases Reported by Dr. H. Douglas Stephens.

W.M., June 30, 1926, six years. Spastic diplegia. Right Royle operation done; later, left side done. Hydrocephalic appearance and looks mentally deficient. Arms and legs spastic with adduction of thighs and urinary incontinence. Was shocked while pulling on the sympathetic cord; recovered; some hæmorrhage from a vein gave trouble. Three days later, right leg warmer than left, less spastic definitely, and knee jerk less exaggerated than left. Six days later still slightly warmer right leg, but now it is just as spastic as the left. Discharged June 12, 1926. Readmitted September 15, 1926. Six years and eight months, paralysed since birth; seven months, premature, never walked, always big head. Birth weight two and a half pounds. Royle operation left lower limb. Discharged September 27, 1926, spasticity unaltered.

J.F., six years, November 7, 1926. Spastic diplegia, difficult birth, paralysed since birth. Began to walk at two years, walked November 20, 1926, and does not appear to be altered. Spasticity unchanged after right Royle operation on leg on November 8, 1926.

W.McF., November 28, 1926, six years and one month. Spastic diplegia. At one year weakened left leg, later both legs, with arms also and difficulty in holding up head. Mental (?), but said to be normal. Incontinence of urine and fæces. Speech bad, unable to stand. December 2, 1926, right Royle operation done. No difficulty. Right leg and foot warmer than left next day, but spasticity as before. Died on December 5, 1926. *Post mortem* examination failed to account for death.

D.H., two years and four months, May 20, 1928. Spastic diplegia. Had Royle operation done, left side, October 14, 1927, November 11, 1927. Mother says child much better since last operation, talking a little, and can sit up. Holds objects better, slides along on back—tendency to microcephaly. Right ramisectomy done May 21, 1928; operation all right. Next day all right, no flushing of limb, and on May 26, 1928, well. Discharged June 1, 1928.

R.L., seven and a half years. Right hemiplegia, said to follow after a fall at seven months of age. Mental defect—five other children a. and w. Had previously had Royle and transplantation of tendons done by me on lower right limb. Operation March, 1926, on right arm; Royle done. Slight erythema, muscles lax, right pupil more than left next day. Slight improvement, active movements noted ten days later.

J.D., five years and one month. July 4, 1926. Spastic paraplegia and stiff joints since three years of age, after attack of vomiting; easy confinement. Another boy has similar gait, but worse than this one. Left Royle done November 26, 1925, and was better than right, and slightly flushed, movements slightly freer in all directions, especially in flexion of left knee, and abduction and adduction of hip. Temperature of both legs equal apparently in three days. On December 5, 1925, no vasomotor changes noted. No difference in muscle tone between two sides, but muscle control slightly greater on left than right, and range slightly greater. Similar on December 11, 1925, December 17, 1925. December 12, 1925, marked difficulty in walking. Complained of pain in region of knee (left) and it seems stiffer than the right one, as if less power and loss of control in left leg. Reflexes similar both sides. Sent to O.P. January 22, 1926, for continuation of massage *et cetera*.

J.P., thirteen years. Spastic paraplegia. At eleven and a half years Royle operation done, left side, and later, T.A. lengthened. At twelve years did Royle operation right side. Practically no improvement in spasticity, marked adductor spasm, left more than right. Stoeffel done left side (crushing obt. N. in pelvis). Discharged after twelve days, walking with much help. Adductors paralysed. July 4, 1926, right Royle operation done; before operation no difference much between limbs *re* spasticity. July 15, 1926, discharged; no detectable difference in temperature or spasticity. November 14, 1926, thirteen years and eight months. Has had Royle operation done on both sides, now for R.T.A. shortening.

F.B., aged eight years and three months. Seen on various occasions with a very gross spastic paraplegia. Said to have walked at one year, but at eighteen months had measles and had been unable to walk properly since; with very great effort some walking is possible. There is much spasm of the adductors and calf groups. On December 17, 1931, left ramisectomy was performed. From this he recovered quite well, but developed diphtheria and was sent to Fairfield Hospital. On readmission a right ramisectomy was done on February 19, 1932. Apart from flushing of the leg, lasting for about ten days, no difference was noted in the plastic tone of the limb. Tenotomy was performed on both sides on March 10, 1932, and his foot placed in plaster. On account of the unhealthy condition of his tonsils, they were subsequently removed, and on May 19, 1932, tenotomy of the *adductor longus* on each side was done. On June 19 he was able to push a chair along the ward, but his gait was by no means satisfactory. Although subnormal mentally and somewhat emotional, he is capable of appreciating reeducative exercises and may improve as time goes on.

Appendix C.

Cases Reported on by Dr. Rupert Downes.

H.B.M., male, aged seven (January, 1928). January 17, 1928, left hemiplegia, noticed at six months. Fits at two to six years of age. Talked before two years. Operation, spastic left hemiplegia. *Talipes equinus*. Babinski. Walks on toes. Upper limb in position of flexed elbow, pronation, flexed wrist, fingers adducted; makes little use of it. Poor mentality. January 19, 1928, left lumbar ramisectomy and division of sympathetic cord. Tenotomy of *tendo Achillis*. January 20, 1928, temperature left popliteal space one and a half inches higher than right. January 29, 1928, more movement noted in left leg. October, 1928, left cervical ramisectomy.

Report from parents, December, 1931: Great improvement in leg, so that it is nearly normal. No improvement in arm.

A.T., ten years and three months. December 17, 1930. Left hemiplegia spastic, ? whether since birth or two years old. Claw-hand and *equino-varus*, both spastic. Mentality good. Operation February 17, 1931. Ramisectomy and division of sympathetic cord. Leg warmer after operation. No improvement at discharge two weeks later. Not seen since.

M.B., five years. February 18, 1929. Spastic diplegia, legs much more than arms. Small tendency to scissor deformity. Mentality deficient. February 19, 1929, left lumbar ramisectomy and cord division. Temperature of left popliteal space 1·6° higher ten hours later; 2° one month later. May, 1932, slightly less rigidity and less prolongation of knee jerk. Improvement slight to nil.

N.M., seven years. September 10, 1924. Spastic paraplegia. Scissor legged; inability to straighten knees or ankles. Hydrocephalus. Mentality very fair. September, 1924: right ramisectomy. Temperature raised immediately. August, 1925: At mother's desire, owing to her opinion of improvement, left ramisectomy. December, 1931, seen by Coates and Downes. Can ride bicycle and at School of Mines (fourteen years of age). Has improved, though still very spastic. Adduction of thighs, not much inconvenience. Can walk, spastic dragging of toes. Double *hallux valgus* and flat foot. Mother thinks improvement definite.

Memo.—Sir R. R. Stawell suggests that in this case vascular dilatation and encouragement might play a part. Mr. Downes to be asked for report on this case in detail later.

Appendix D.

Cases Reported by Dr. Hugh Trumble.

M.P., aged twelve. S.I.P., September 28, 1927–October 15, 1927. Spastic paraplegia. Spastic paralysis since age

of three. Walked very badly. Plantars extensor. Mentality fair only. Operation September 29, 1927, left lumbar ramisection. Royle operation. Temperature changes well seen. There was no obvious improvement following operation.

A.D., aged nineteen. S.I.P., October 6, 1930–November 11, 1930. Spastic diplegia with gross circulatory disturbance of both legs. History: Spasticity both legs since birth. Walked with difficulty. Reflexes legs marked by spasticity. Extensor plantars. Circulation of legs poor (blue, mottled and cold). Wassermann negative. Operation, October 14, 1930, left-sided ramisection of Royle type. Immediately left limb hotter than right. Later, *tendo Achillis* lengthened both sides. Reported March 26, 1931. Left leg warm and comfortable. Right leg chilblains *et cetera*. Walks better, probably on account of tendon lengthening.

Note.—The operation was performed mainly for the relief of "chilblain circulation of severe type". This was largely relieved.

H.D., aged twenty. S.I.P., July 31, 1927–August 29, 1927. Post-encephalitic *paralysis agitans*. Influenza twenty-two months before operation. Developed *paralysis agitans*. All limbs spastic. Right greater than left. Tremor exaggerated. Operation, August 8, 1927, right-sided cervical ramisection, during which cervical sympathetic cord was injured. Temperature rose right arm. Spasticity not affected. Tremor not affected, soon after operation.

B.A., aged sixteen. Spastic triplegia. S.I.P., May 30, 1927–August 24, 1927. At age of six years was normal, then developed paralysis. ? syphilitic. The Wassermann now negative. Gross spasticity left arm and both legs. Mentality not good. Operation, June 9, 1927, left lumbar ramisection. June 27, 1927, right lumbar ramisection. Temperature changes well seen. Type of knee jerks unchanged. Spasticity seems a little less for a while. State of patient no better as regards walking *et cetera*.

Appendix E.

Reports from Dr. C. W. B. Littlejohn.

I have done the operation of ramisectomy on twelve cases of spastic paralysis, the earliest in 1923, N.J., at the Children's Hospital. Seven cases were done in England. In none of the cases has there been any lasting benefit, although in several there has been an apparent relaxation of the spasticity. In certain cases this release has been appreciated more by the patient than by the observer. The cases done in England were examined by several prominent physicians and physiologists, but, apart from vasomotor changes, they could find no clinical change in the condition of the limbs.

(*Signed*) C. W. B. LITTLEJOHN.

June 10, 1932.

Appendix F.

Cases Reported by Sir Richard Stawell.

Though I have been associated as an observer and as a consultant concerning several hospital patients on whom ramisectomy has been performed, I have personally recommended the operation in only two patients.

First: Miss M.S., aged thirty-seven years. Seen first on August 14, 1924. Operating surgeons, Messrs. Kilvington and Hailes. Date of operation, October 22, 1924.

Clinical history: Was apparently well until about twenty years of age, then there occurred acutely some loss of power in right arm and right leg. This loss of power progressed and was associated with stiffness of these limbs. No clear history could be obtained of the actual mode of onset or of associated signs and symptoms of this original illness, which occurred when patient was resident in the country. In July, 1924, the father had communicated by letter with the late Professor Hunter in reference to this patient. Professor Hunter wrote that, as far as he could judge, he considered it a suitable case for ramisectomy, but referred the patient to me. On examination there were signs of right hemiplegia with rigidity, contracture and some constantly recurring spasm, but with some power of movement. On the right side (i) the knee jerks were very active with a tendency to rigidity and "maintenance of posture", (ii) ankle clonus was present, and (iii) the plantar reflex was "extensor" in type. The right forearm, when at rest, was kept in position of semiflexion with pronation of the forearm. In spite of this, voluntary movement was possible. There was no loss of any form of sensation. There was no evidence of changes about the optic disks. There was no history of headaches. The patient, who was of ordinary intelligence, stated that when she was "not trying" she could walk fairly well, but directly she tried to improve her movements exhausting spasmodic contractions occurred. After consultation with Mr. Kilvington, Dr. Royle's operation was considered justifiable. Cinematograph films of the patient's method of walking and of the range and type of the right and left knee jerks were taken. On October 27, 1924, the operation of right-sided lumbar ramisectomy was performed by a lumbar incision. The pieces of rami removed were fixed, stained and microscopically examined. Immediately after the operation the right leg was warmer than the left. Four months later (February, 1925) the patient was seen by me again. She reported "no appreciable improvement", though I thought at that time that there was slightly lessened "spacticity", but the patient explained that this varied and was "usually as bad as ever" and that she got worn out with dragging her foot and forcing her right leg forwards.

In July, 1931 (nearly seven years after the operation), the father replied, in answer to a letter from me, that "there has been no noticeable improvement in my daughter's condition except that her right foot is a little warmer".

Second: Miss H.B., aged thirty-seven years in 1923, was seen by me first on November 23, 1923, within a week of the medical congress of that year, at which Dr. Royle and the late Professor Hunter had presented their work on the operation of sympathetic ramisection as a method of relief for "spastic" conditions of the limbs.

The history of this patient was that when seventeen years of age she got a backache with stiffness of the neck and headache, followed within ten days by signs of a partial transverse myelitis involving the level of the ninth dorsal segment of the cord. Two to three months later, a surgeon in a country town performed a laminectomy, but nothing definite was found (personal communication from the surgeon). Eighteen months later she was able to stand, all numbness had gradually disappeared, but her legs were so rigid she could hardly move them. On examination in 1923 she had a condition of spastic paraplegia, slightly more marked on the left side, and she had loss of sense of posture and of passive movement in the toes and feet, more marked on the left side. Even at that date the question of sympathetic ramisection was considered, but it was felt that further observations on later and lasting results were necessary before recommending the operation. It was felt that the immediate effects of the operation in apparently relieving spasticity might not be altogether due to cutting off the sympathetic nerve supply to the limbs, but that, in the very nature of things, some emotional effect must have been produced, which, as in all forms of mental and physical activities and disabilities, would have a temporary favourable influence.

Later on, in 1923, radiological examination of the spinal column and the routine examination of the cerebro-spinal fluid revealed nothing abnormal, and, some years later, by the use of lipiodol, it was shown that there was no block or narrowing within the spinal canal.

By reeducation methods some improvement occurred, so that walking was not so difficult. The improvement was not marked, and after four years of reeducation and because of some evidence, apparently in favour of ramisectomy, I consented to the operation. This was carried out on December 5, 1927. At first, following the operation we thought there was some lessening of the spasticity. There was certainly increased warmth about the lower limbs. I had the opportunity of observing this patient for three years after the operation and I could find no lessening of spasticity and no sustained improvement which could be attributed to ramisection. The patient has since reported to me by letter that she is "just the same".

Appendix G.

Report by Dr. W. Allan Hailes.

Dr. Hailes reported (June 4, 1931) that he had operated upon two cases of Little's disease and that, although thermal changes were noted, no improvement in the condition of spasticity took place.

British Medical Association News.

SCIENTIFIC.

A meeting of the Section of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association was held in the Robert H. Todd Assembly Hall, British Medical Association House, Sydney, on November 21, 1933, Dr. S. E. Jones in the chair.

The Problem of Mentally Defective Children in New South Wales.

Dr. A. E. Machin read a paper entitled: "The Problem of Mentally Defective Children in New South Wales from the Educational and Vocational Points of View" (see page 370).

Dr. G. E. Phillips, of the Special School, Glenfield, opened the discussion. He first thanked Dr. Machin for his kind remarks about the Special School at Glenfield. Regarding the cost of the education of mental defectives, the problem concerned all those present as taxpayers. In the previous year the cost of each child at Glenfield was £86, as against £13 or £14 per child in the ordinary schools, but £26 to £30 were returned in fees. When the number of mentally defective children in the State was considered, the magnitude of the problem would be realized. He was concerned mainly with education of backward children and aimed at taking those who could be returned to the community at large with no other supervision than the parents could exercise. That was why the Glenfield Special School was limited as a general rule to children whose intelligence quotients were not below 55%. The number of girls was only about a third that of boys, and in their case the problem was not nearly so complex, as the majority would return to their mothers for home duties. Very few girls attempted to earn their living outside the home. Very few of the children failed to reach the educational standard of reading newspapers, writing intelligible letters and making change. Of the boys sent out from the school, 62% had been in actual employment when a survey was made two months previously. All who had left the school, except one, had had employment, and this one boy was a sufferer from chorea who was still continuing his physical education. Sixty-two *per centum* represented a very satisfactory result considering the prevailing economic conditions—probably as good as that for normal boys. The majority were on the land; others were employed in repetitive work in factories, as messengers and so on.

His own problem as far as education was concerned was the lop-sided child, whose intellectual and emotional development had not kept pace. One boy out of school was supernormal in common sense and nous; he had an intelligence quotient of only 44% and after six years at school was unable to make any progress beyond the second standard and could only write a very poor letter, but outside of school life he was really equal to a boy of fifteen years. They were apt to identify common sense and intelligence, but to Dr. Phillips's mind this was not correct. Others could do school work very well, but outside school were hopeless. One boy of twelve years could not dress himself, but he was living in a home with seven women with no men, and they insisted on doing everything for him. This boy was in third class at school, but outside was helpless. It seemed that common sense was largely a matter of emotion and called into play something a little different from intelligence. Everyday happenings rather depended on the way things struck them—a certain solution felt to be right and followed. This problem struck Dr. Phillips very forcibly at Glenfield; there must be some lack of relationship between intelligence and common sense or nous. When asked if such a child—the emotionally retarded child—was likely to be able to earn his living, he could not provide a satisfactory answer. When there was a considerable disparity between emotional and intellectual development, the most complex and sometimes insoluble problem was presented to the educationalist. The boy of intelligence quotient 44% was certainly going to be able to earn his living, as for one thing he was very capable with animals. The lower limit of educability was placed at intelligence quotient 50%, but on the other hand, he had had some remarkable successes with boys of intelligence quotient below 50%. In the case of E.S., his schoolmaster had reported that he could do no schoolwork whatever, but he knew when the other boys were "pulling his leg". He had been accepted for Glenfield on this recommendation, although his intelligence quotient was below 50%, and he learned to do farming operations and how to drive a motor lorry. He reached an educational standard in two and a half years which usually took boys four years to attain.

Dr. Phillips finally assured the members present of a most hearty welcome if they should desire to visit Glenfield at any time.

Professor W. S. Dawson said that first of all he would like to thank Dr. Machin for bringing this very important question before the meeting in such an interesting manner. Dr. Machin had very fully covered the ground and had brought forward problems which would have to be met before long. Professor Dawson could not quite understand why special classes were a failure; that was a very important question because of the cost of educating mentally defective children in special institutions. Such classes had operated with considerable success in other parts of the world. Quite recently he had had the opportunity of speaking with Dr. Todd, a medical officer of the Education Department of New Zealand, who said that there were no less than seven special classes in Auckland. A great deal seemed to depend on the selection of the right type of children, and such classes often proved a failure because they were just used as a dumping ground for scholastic misfits. If children were admitted with an intelligence quotient of 75% or a little above, and if they did not display too many temperamental disabilities, and also if the numbers were not too great, valuable work could be done. Special institutions were extremely expensive, and, as Dr. Phillips had pointed out, it did not seem likely that there would be much development in that direction in New South Wales for a considerable time. When he had visited Glenfield he had been much impressed with the work being done, but at the same time he thought that an institution must be larger than Glenfield to be worked economically. Glenfield might expand in the future and he considered that to provide the greatest variety of educational training, recreation and character development at the least expense per individual, it should be at least six times its present size.

He then said that he would like to refer to some figures representative of an average English state school population. Some 12% were classified as subnormal, and these were children with an intelligence quotient below 80%. The dull and backward numbered 8%, and definitely feeble-minded about 1%. It was the dull and backward type of 75% to 85% intelligence quotient who really deserved most attention. He would suggest that, instead of further attention being given to the building of institutions, special classes would give more value to the State for the money expended.

Education in special classes and institutions was apt to be of considerably less value, unless there was a good system of social supervision after the educational period was completed, and such a system might well be developed in New South Wales. In England various agencies did quite a lot of work for the children after they left school. Training would not reap its due reward without an adequate follow-up system.

Dr. S. E. Jones said that he would like to thank Dr. Machin for his excellent paper, which was so interesting to psychiatrists, and to thank Dr. Phillips, from Glenfield,

for taking part in the meeting. Most of the members had had the pleasure of visiting Glenfield. He quite agreed with Dr. Phillips that there was a lack of coordination in the various bodies in looking after the individual. Referring to the psychology of the backward children, as mentioned by Dr. Phillips, Dr. Jones stated that there was a failure in emotional development; that hitherto in educational systems over much attention was paid to intelligence and not enough to the emotional training of children.

In replying, Dr. Machin thanked the speakers for their appreciative remarks. In reference to Professor Dawson's views about special classes, he had not wished to convey the impression that special classes were altogether a failure, but by themselves they were not a solution of the whole problem. Special classes might be used in certain districts for certain types of children. They had not had sufficient experience in New South Wales to condemn them altogether. Backward children, roughly those with intelligence quotients ranging from 75% to 85%, were much more numerous than mental defectives and represented a much bigger problem from the teacher's point of view than mental defectives. He would suggest that such children might suitably be trained in special classes. The 12% classified as subnormal in the English figures quoted by Professor Dawson were a very different matter to the 1% of mental defectives mentioned in Dr. Machin's paper. If the number of children with intelligence quotients up to 85% were estimated, the figure would probably be much the same for New South Wales. Subnormal was defined as anything below normal intelligence. Dr. Machin considered 1% to be a conservative estimate of the incidence of mental deficiency in New South Wales.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Lloyd-Green, Lorna, M.B., B.S., 1933 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

Drury, Harrie Denison, M.B., B.S., 1933 (Univ. Melbourne), Alfred Hospital, Prahran, S.1.

Medical Practice.

LIQUID EXTRACT OF ERGOT.

THE following note has been received from the Secretary of the Pharmaceutical Society of Victoria with a request for publication.

℞

Pot. Brom.	gr. x.
Ext. Ergot. Liq.	m. xx.
Ext. Glycyrrh. Liq. ..	ʒ ss.
Aq. ad	℥ ss.

Mitte ℥ vi

℥ ss *ex. aq. nocte.*

This prescription was presented to a suburban pharmacist recently. Acting on information supplied in current literature, the dispenser sent out the ergot in a separate bottle, labelled "Twenty drops with each dose of mixture".

Later on the customer called and said that she got the same mixture on another prescription from a city chemist in a single bottle. On telephoning the chemist who dispensed the prescription for the first time, it was ascertained that he used the B.P. 1914 ergot, as the 1932 preparation was incompatible.

In asking for some official direction, the second chemist said that apparently the prescription could be made up as follows:

1. Use B.P. 1932 Liq. Ergot. in separate bottle.
2. Use B.P. 1914 Liq. Ergot. in one bottle.
3. Use B.P. 1932 Liq. Ergot. in one bottle, trusting that the patient will get a mixture that will keep for more than twenty-four hours.

Dr. Byron L. Stanton, M.R.C.P., Ph.C., to whom the question was referred, states that:

1. The only drug of this name since the adoption of the B.P. 1932 is that defined by the B.P. 1932, consequently to dispense the B.P. 1914 preparation (unless specifically ordered by the physician) is an error.
2. Reports and observations show that the B.P. 1932 preparation deteriorates very rapidly when mixed with water, that is, when its alcohol content is reduced.
3. It should, therefore, not be dispensed in mixtures, but given separately with an exact measure. (The term "drops" should be avoided.)
4. Under the circumstances the second chemist followed the correct procedure in dispensing the ergot separately.
5. In dispensing separately the chemist should (*a*) notify the doctor of the procedure adopted and (*b*) note on the prescription the method of dispensing adopted.

The early publication of an addendum to the B.P. 1932 is forecast by the British Pharmacopœia Commission, and it is probable that the difficulty that has arisen in dispensing mixtures containing ergot may be overcome by the adoption of a revised standard. In the meantime the procedure suggested by Dr. Stanton should be followed.

Research.

SCHOLARSHIPS IN BLOOD DISEASES.

THE Commonwealth Department of Health has received advice from the Office of the High Commissioner in London that the Trustees of the Lady Tata Memorial are offering four scholarships for research in blood diseases, with special reference to leuchæmias.

These scholarships, which are similar to four awarded in June, 1933, are open for award in June, 1934. They are each valued at £400 and are tenable for one year from October 1, 1934, and renewable to a normal maximum of three years. They are available for both sexes and will be ordinarily awarded on a whole-time basis, but the appointees may hold part-time teaching posts if, in the opinion of the trustees, their duties will not prevent the devotion of their chief interests to the specified research.

Applications for the scholarships must reach the Secretary, Lady Tata Memorial Trust, Capel House, 62, New Broad Street, London, E.C.2, not later than April 15, 1934, or up to April 30 if delayed by special circumstances.

Candidates must advise name, age, sex, nationality, qualifications, proposed line of research, methods to be used, institute or laboratory in which research is to be done, and the name of the director who will supervise it. Testimonials, in confidential letters addressed to the Secretary, should be sent at the same time by not more than three persons able to speak from personal knowledge of the candidate's character, ability and experience.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on March 5, 1934.

The Diploma in Public Health was awarded to Frederick William Arthur Clements, M.B., B.S. (Sydney).

The degree of Doctor of Veterinary Science was conferred *in absentia* upon Harold Roy Carne, B.V.Sc., for his thesis entitled "Studies on Corynebacterial Infections of Domestic Animals". The examiners reported that the thesis was a contrbution of distinguished merit, adding to the knowledge and understanding of the subject matter. (Dr. Carne, who is Lecturer in Veterinary Pathology and Bacteriology in the University of Sydney, graduated Bachelor of Veterinary Science in 1923 with First Class Honours and the University Medal. He is at present in England continuing his studies and investigations as the holder of a Rockefeller Fellowship awarded to him last year.)

The Walter and Eliza Hall Travelling Fellowship in Veterinary Science was awarded to Mr. D. F. Stewart, B.V.Sc. (Mr. Stewart graduated in 1932 with Second Class Honours, and proposes to undertake research work in mastitis at the London Veterinary Research Institute and at the Institute of Comparative Pathology, Cambridge University.)

The Pharmaceutical Association of Australia and New Zealand forwarded a contribution of £25 for research work in the Department of Pharmacy in recognition of assistance rendered by Mr. H. Finnemore, Lecturer in Pharmacy. The gift was accepted with grateful thanks.

The matriculation ceremony will take place on Tuesday, April 19, 1934, at 4 p.m., when all matriculants will be required to attend.

The annual conferring of degrees has been arranged for Saturday, May 5, for the Faculty of Arts, and for Saturday, May 12, for all other faculties.

It was decided that the quinquennial election of Fellows of the Senate should be held on Monday, November 19, 1934.

The following appointments were approved: Dr. A. W. Morrow as Honorary Demonstrator in Pathology; Mr. D. S. R. Abbott as Assistant House Surgeon in the School of Veterinary Science; Dr. W. I. T. Hotten as Lecturer in Anæsthetics; Dr. E. S. Holloway, Dr. Hugh Hunter and Dr. W. I. T. Hotten as Tutors in Anæsthetics; Mr. R. H. Parker, M.Sc., as half-time Demonstrator in the Department of Organic Chemistry; Mr. N. E. West, Mr. E. R. Cole and Mr. A. N. Jaffray as Demonstrators in the Department of Pharmacy.

Correspondence.

A REQUEST.

SIR: I would be very grateful for the assistance of anyone who has observed the effects produced by misplaced buried teeth (aberrant teeth) and impacted teeth (not teeth in dermoids), teeth in unusual situations, in helping me gather material for an article on this subject, which will be written in collaboration with L. Monier, M.D., D.S., Paris.

Would appreciate any information sent to me bearing on the below listed points, and for any reprints, clippings or references to articles or books dealing with the same. *A.* At what age and how was the buried, aberrant or impacted tooth or teeth discovered? *B.* At what age did the removal take place? *C.* Has malignancy been observed to precede or follow the removal or failure to remove any such tooth or teeth? *D.* What signs, symptoms or effects (local, general or special) drew attention to such tooth or teeth? *E.* What method was used to remove the same? *F.* What anæsthetic was used? *G.* What effect, if any, did the removal have upon the previously mentioned signs, symptoms or effects? *H.* Did the removal have any effect or complications other than those previously mentioned? *I.* The race of the patient. *J.* The sex of the patient. *K.* The position of the tooth or teeth.

Any prints of X ray films or plates, or any diagrams or drawings showing the position and relation to other teeth, and other structures will be valued and appreciated. These will be returned when requested, and credit will be given for any material used, so please write name and address plainly.

Yours, etc.,

ALONZO MILTON NODINE,
D.D.S. (N.Y. Univ.), L.D.S., R.C.S. (Eng.), L.R.C.P (Lond), M.R.C.S. (Eng.).

117, The Ridgway,
Wimbledon,
London, S.W. 19,
England.

Undated.

Post Scriptum.—Replies may be in English, French, Spanish, German, Italian and Russian.

CALCIUM AND PHOSPHORUS METABOLISM.

SIR: I think Dr. Hansman and Dr. Wilson, whose papers have recently appeared in your journal, are to be congratulated on the excellent work they have undertaken in this direction. This matter was of a great deal of interest to me twenty years ago, and the results of modern research have largely confirmed the views arrived at at that time. There are one or two points which may be of interest to bring forward and give food for thought. We know that in the matter of the mineral constituents of the blood the body allows of very little variation. A little too much or too little is dealt with at once, so unless the mechanism for regulating these delicate adjustments has been broken down or seriously interfered with we shall see very little change. We are in the habit of being taught certain things, believing them to be true and taking them for granted, often without any thought on the matter ourselves. Take the statement that: "It is now generally agreed that all food calcium is converted into calcium chloride in the alimentary tract and absorbed as such." Take it for granted that all food calcium is converted into calcium chloride in the stomach, but for how long does it remain as a chloride once it passes into the alkaline duodenal secretion? We can ignore any absorption of calcium chloride through the stomach wall. Has calcium chloride been demonstrated in the jejunal contents? It is stated over and over again that the bowel secretes calcium. Who is to say how much of this has ever been absorbed? We have to consider two alternatives. One is that all the calcium is converted into and absorbed as calcium chloride and then excreted by bowel and kidneys; the other, that the chloride may be converted into some insoluble compound, such as phosphates or soaps, which renders absorption extremely difficult, leaving calcium salts which have never been absorbed to be passed out in the fæces. In human beings with an acid urine there is sufficient acid to insure the calcium being excreted by the kidneys; in animals with an alkaline urine this is not so and excretion is largely through the bowel.

In favour of this latter contention clinical observation has much to say. In the average individual on the ordinary diet it is assumed that there is ample calcium for all requirements, yet when an easily assimilated form of calcium is given, such as the lactate, marked changes take place. This is well recognized and observed so frequently that it cannot be denied. It is also well recognized that children at times pass white stools consisting of soaps in which calcium is combined, these children showing all the signs of rickets, presumably through their inability to utilize the calcium. The action of the pancreatic secretion on fats is extremely rapid. The fatty acids combine with the calcium, potassium and magnesium salts to form soaps, so there is a very short period of time in which calcium can be absorbed as the chloride. The solubility of soaps varies according to the type of fat split up. It is known that the soap formed from cod liver oil is one of the most soluble. Bile plays a large part in the breaking down of soaps, and with this in view, some years ago I employed pig and ox bile in the treatment of goitre with a certain amount of success. The value of cod liver oil in the absorption of calcium from the bowel is well known, also that of irradiated ergosterol. Is the absorptive value of cod liver oil due to its forming an easily soluble soap or not? If not, what is its relation to irradiated ergosterol? In operative tetany it is the experience of everybody to find how little good calcium salts are by the mouth, yet when vitamin *D* is supplied in sufficient quantities, absorption takes place. The vulnerable point is the bowel wall and that the vitamin *D* has rendered the calcium soap capable of absorption or it has acted in some way on the liver to supply sufficient bile salts to break down the soaps. It would be extremely interesting to know exactly in what way vitamin *D* acts. On page 83 Hansman quotes from Bauer, Marble and Claffin: "The administration of five milligrammes of irradiated ergosterol a day on an individual with parathyroid tetany receiving a high calcium intake produces the following changes: (*a*) A decreased fæcal calcium excretion. (*b*) An increased urinary calcium excretion. (*c*) An increased

positive calcium balance. (*d*) A rise in serum calcium . . ."

The decreased fæcal excretion and increased urinary secretion point to the absorption and utilization of calcium within the body, but there is nothing to tell us that any of the fæcal calcium has ever been absorbed.

Of two facts we are quite sure. One that the function of the parathyreoids is to draw calcium from the bones, which are the great storehouse of that commodity, and the other that vitamin *D* or irradiated ergosterol causes absorption from the bowel and deposits it in the bones. All iodine has to pass through the thyreoid before it is properly metabolized for use as thyroxin. Has all the calcium to be first deposited in the bones before it is fit to be employed in the repair of tissues and other functions? There may be much in this.

In the case of calcium depletion over a long period it is improbable that one would expect to find great changes in the blood calcium, any more than there would be of sodium chloride if deprived of that commodity. I do not know at what limit of depletion of calcium the body is capable of functioning, but I presume it has been worked out. In the case of long depletion with adequate absorption the storehouse will again be filled, but once the blood calcium has reached its normal level it will remain at that, and any surplus got rid of by the kidneys or possibly bowel, or not be absorbed, as absorption would not come into play unless needed. Now should the key to the storehouse be mislayed, as in hypoparathyreoidism, although the calcium is normal in the serum, no more will be available from the bones. With hyperthyreoidism and hyperparathyreoidism together both the storehouse and the tissues will soon be emptied, as seen in thyreotoxic goitre. All the organs of our body are under automatic control; no matter what we do, we cannot regulate their function. They are handled by the two great involuntary nervous systems and their associating hormones. We like to think that dysfunction of the thyreoid with goitre may be due to some interference with the metabolism of calcium and iodine. By no manipulation of one or the other of these have we succeeded in producing anything resembling a goitre. If goitre was due to some metabolic disturbance we should get some consistency in research, but instead we find the most bewildering state of affairs. If goitre was the result of a direct infection of the gland with some organism we would expect to get such a bewildering state of affairs, because no two infections would be quite the same. With a generalized infection of the gland it would be unreasonable to expect the parathyreoids to be overlooked. It is interesting to realize that whilst the thyreoid is associated with the sympathetic group, the parathyreoids are associated with the vagal or parasympathetic group. This suggests that they are in some measure antagonistic in their action, but combined in one service. We know that the parathyreoids steal the lime from the bones, and we know that with hyperthyreoidism there is a great excretion of lime, the one supplying, the other breaking down.

If we accept the fact that the thyreoid is invaded by organisms in thyreotoxic goitre, what is likely to happen? If we take analogy from other sources we should expect great interference with normal function, we should expect great hyperactivity due to irritation. Not only would this happen to the thyreoid, but to the parathyreoids embedded in it. It is then not so difficult to see the train of symptoms and findings which are so prominent in this complaint. There must of a necessity be a mechanism that regulates the amount of calcium circulating in the blood serum. I should like to suggest that this balance is kept between the thyreoid and parathyreoids. There are two good reasons for this conjecture. In myxœdema, where there is very little thyroxin available and an adequate calcium supply, after the storehouse of the bones has been filled there is an overflow into the tissues, resulting in a saturation of the tissues with colloid. This colloid, as has been expressed in previous papers, shows every evidence of excess calcium. The administration of iodine or thyreoid extract brings this into solution. The second reason is that there is a generally recognized excessive outpouring of calcium in hyperthyreoidism. In the two cases, Edith A. and Emma B., with hyperthyreoidism and hypoparathyreoidism, under these conditions they could show no excessive excretion because the storehouse was not unlocked by the parathyreoids. These two cases also suggest that whereas the irradiated ergosterol was causing absorption of calcium and storing it in the bones, owing to the hypofunction of the parathyreoids very little was available for utilization, as only a slight calcium balance was obtained. Once the storehouse was full, the automatic mechanism would cease to absorb more, in spite of the irradiated ergosterol, leaving the calcium balance little unchanged.

Yours, etc.,

S. PERN.

12, Collins Street,
Melbourne,
February 14, 1934.

THE TREATMENT OF RODENT ULCER.

SIR: In his paper at the Tasmanian congress Dr. Clendinnen states that radium should be the only method of treatment for rodent ulcer. Seeing so many failures of radium at the Melbourne Hospital and in private may give me a jaundiced view of radium. Its advantages are its simplicity and ease of application; its disadvantages are not a few: (i) Pain after application; (ii) production of a painful innocent ulcer which refuses to heal; (iii) when it does not cure it often produces a very malignant type of ulcer; (iv) the appearance of multiple rodents in quite large numbers (I have seen twelve ulcers in the one patient); (v) that some radium users persist in applying radium for years. I believe that some experts maintain that if radium does not cure at the first application it seldom succeeds at the second and should never be applied a third time.

Radium has no advantage over electro-surgery in simplicity and ease of application. A local anæsthetic, a few seconds of treatment, and a sear equal to radium should be the result.

Has anyone ever seen multiple rodents in a case treated only by electro-surgery? Of course, there may be no truth in the surmise that radium, while abolishing the ulcer in one place, produces it at another, and it may also be true that many of the failures of radium are due to the unintelligent use of radium. I certainly have seen two cases of multiple rodents who have had no treatment at all.

Dr. Clendinnen notes in cancer of the tongue a remarkable tendency to recurrence after using radium in areas quite remote from the original site. I have never seen this occur after electro-surgery, but my cases have been too few to be of use in considering this question. It is useful support, however, to those who maintain the possibility of radium producing cancerous lesions. I thought most surgeons agreed that radium had no place in the treatment of rodent ulcer when it had invaded bone or cartilage. The agony produced by radium where bone is involved should deter all but the most prejudiced from using it.

As regards cancer of the lip, I also contend that electro-surgery is preferable to radium.

Yours, etc.,

W. KENT HUGHES.

22, Collins Street,
Melbourne,
February 17, 1934.

THE TONGUE OF THE TRAVELLER: A PROTEST.

SIR: I would like to sound a note of warning to those medical men who submit to a newspaper interview on returning to their homeland, after sitting at the feet of the wise men of the Old World. In many cases, their exuberance at once more setting foot on Australian soil, overcomes that discretion which distinguishes our medical brethren. They burst forth into panegyrics of the wonders they have seen, and by comparisons, which are proverbially odious, tend to wither that frail plant—our medical pres-

tige. The results of the interviews invariably appear under startling captions and the avid reporters generally add a little for good measure.

As Western Australia is, as a rule, the first State to welcome them, its newspapers are the first to proclaim our medical, surgical, and anæsthetic backwardness.

I would through your columns ask each medico returning from his Odyssey to remember the old aphorism: "The ox has a large tongue and he cannot speak. Man has a small tongue and he must not speak."

Yours, etc.,
M. K. Moss,
President, Western Australian Branch,
British Medical Association.

Perth,
February 22, 1934.

Obituary.

PERCY OSWALD LORD.

We regret to announce the death of Dr. Percy Oswald Lord, which occurred on March 2, 1934, at Hawthorn, Victoria.

HAROLD BROWNE.

We regret to announce the death of Dr. Harold Browne, which occurred on March 7, 1934, at Edgecliff, New South Wales.

Diary for the Month.

Mar. 19.—Tasmanian Branch, B.M.A.: Council.
Mar. 20.—New South Wales Branch, B.M.A.: Council Quarterly.
Mar. 21.—Western Australian Branch, B.M.A.: Branch.
Mar. 22.—New South Wales Branch, B.M.A.: Annual Meeting.
Mar. 23.—Queensland Branch, B.M.A.: Council.
Mar. 24.—Tasmanian Branch, B.M.A.: Annual Meeting.
Mar. 27.—New South Wales Branch, B.M.A.: Council: Election of Office-Bearers and Appointment of Standing Committees.
Mar. 28.—Victorian Branch, B.M.A.: Council.
Mar. 29.—South Australian Branch, B.M.A.: Branch.
April 3.—Tasmanian Branch, B.M.A.: Council.
April 4.—Western Australian Branch, B.M.A.: Council.
April 5.—South Australian Branch, B.M.A.: Council.
April 6.—Queensland Branch, B.M.A.: Branch.

Medical Appointments.

Dr. F. J. Foley (B.M.A.) has been appointed, pursuant to the provisions of the *Workers' Compensation Act*, 1928, to be a Certifying Medical Practitioner at St. Kilda, Victoria.

* * *

The undermentioned have been appointed Resident Medical Officers to the Adelaide Hospital, South Australia: Dr. J. M. M. Gunson, Dr. P. Stratmann, Dr. B. P. Funder, Dr. J. H. Johnston, Dr. S. Peters, and Dr. R. Hayes.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi, xvii and xix.

Infectious Diseases Hospital, Fairfield, Victoria: Junior Resident Medical Officer.

Launceston Public Hospital, Launceston, Tasmania: Resident Medical Officer (male).

Royal North Shore Hospital of Sydney, New South Wales: Honorary Assistant Surgeon.

The Broken Hill and District Hospital, Broken Hill, New South Wales: Resident Medical Officers.

The Ryde District Soldiers' Memorial Hospital, Ryde, New South Wales: Honorary Medical Officers, Resident Medical Officer.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
South Australian: Secretary, 307, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

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